A COMPARATIVE STUDY OF INTUBATING CONDITIONS BETWEEN PROPOFOL-FENTANYL-MIDAZOLAM AND PROPOFOL -FENTANYL- LIGNOCAINE GROUPS WITHOUT NEUROMUSCULAR BLOCKING AGENTS.

Dissertation submitted in partial fulfillment of
M.D. DEGREE EXAMINATION
M.D. ANAESTHESIOLOGY- BRANCH X
CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU.
APRIL 2013

CERTIFICATE

This is to certify that this dissertation titled **"A COMPARATIVE STUDY OF INTUBATING CONDITIONS BETWEEN PROPOFOL- FENTANYL-MIDAZOLAM AND PROPOFOL - FENTANYL- LIGNOCAINE GROUPS WITHOUT NEUROMUSCULAR BLOCKING AGENTS"** has been prepared by Dr. R.Selvakumar under my supervision in the Department of Anaesthesiology, Chengalpattu Medical College &Hospital, Chengalpattu during the academic period 2010-2013 and is being submitted to The Tami Nadu DR. M. G. R. Medical University, Chennai in partial fulfillment of the University for the award of the Degree of Doctor of Medicine (Branch X-MD Anaesthesiology) and his dissertation is a bonafide work.

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DECLARATION

I, **Dr. R.Selvakumar**, solemnly declare that the dissertation **"A COMPARISON OF INTUBATING CONDITIONS BETWEEN PROPOFOL - FENTANYL - MIDAZOLAM AND PROPOFOL - FENTANYL - LIGNOCAINE GROUPS WITHOUT NEURO MUSCULAR BLOCKING AGENTS"** is a bonafide work done by me in the Department of Anaesthesiology, Chengalpattu Medical College & Hospital, Chengalpattu, after getting approval from the Ethical committee under the able guidance of **Prof. Dr. V.JAYARAMAN M.D.D.A.,** Professor & HOD, Department of Anaesthesiology, Chengalpattu Medical College, Chengalpattu.

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Dated:24.03.2011

Sub: Medical Education – Chengalpattu Medical College, Chengalpattu – Dr.R.Selvakumar, Post Graduate Student in M.D.(Anaesthesiology) – Admitted during the Academic year 2010-2011 – Dissertation submitted for the Approval of the Human Ethical Committee for the study work entitled **"Comparison of Inj.Midazolam and Inj.lidocaine for tracheal intubation without neuromuscular blocking agents"** – Approved - Orders - Issued.

Ref: 1) Application dated: 21.01.2011 of the Individual.
2) Letter dated 21.01.2011 of the Head of the Department of Anaethesiology, Chengalpattu Medical College, Chengalpattu.

...

Dr.R.Selvakumar, Post Graduate Student in M.D.(Anaethesiology) of this college has submitted a Dissertation for the study work entitled "entitled **"Comparison of Inj.Midazolam and Inj.lidocaine for tracheal intubation without neuromuscular blocking agents"**. The study of the above Dissertation Work of the above Individual has been approved by the Human Ethical Committee of the Chengalpattu Medical College, Chengalpattu which is forwarded herewith for further course of action.

Further, as per the Regulations of the Tamil Nadu Dr.M.G.R.Medical University, Chennai – 32 with regard to submission of 4 **Copies of Dissertation** with C.D.(4 copies) for the Post Graduate Degree Course, the Dissertation shall be a bound volume of minimum 50 pages and not exceeding 75 pages of typed matter (Double line spacing and one side only) excluding Certification, acknowledgement, annexure and Bibliography". The name of the candidate should not be found anywhere in the Dissertation except in the Certificate. The Dissertation should be forwarded through the Unit Chief/H.O.D./Head of the Institution as per the Colour (**LIGHT GREEN**) as communicated by the University.

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To

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CHENNAI, TAMILNADU . APRIL 2013

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CONTENTS

INTRODUCTION

Before the early 20th century, tracheal intubation was done for conditions including tumors of the oral cavity and obstruction in the larynx. It was done crudely using fingers as a makeshift laryngoscope and without using any drugs. In 1909, ether anesthesia was introduced for the purpose of tracheal insufflations. In 1913, Rowbotham modified the technique and described a series of cases. These tracheal tubes were wide bore catheters and forceps was used to guide them into the trachea.

Before the development of neuromuscular blocking agents, endotracheal intubation was done under deep inhalational anaesthesia with ether. Following this, halothane was used to facilitate tracheal intubation. Nowadays, sevoflurane is the most commonly used drug for inhalational induction in peadiateric age group. In 1942, neuromuscular blocking drugs were first introduced into clinical practice to facilitate tracheal intubation.

In 1948 Lewis et al used thiopentone sodium for tracheal intubation without using neuromuscular blocking drugs. His study showed that adequate intubating conditions were achieved using thiopentone sodium alone. Tracheal intubation is usually done with muscle relaxants supplemented with induction agents. Over the past few years, several factors have led the researchers to consider omitting

neuromuscular blocking agents for tracheal intubation. Endotracheal intubation was facilitated by the apparent ability of propofol to blunt responses to tracheal stimulation and the availability of the short acting opioids, remifentanil and alfentanil.

Tracheal Intubation without the use of neuromuscular blocking drugs were used to assess the airway by laryngoscopy and to ascertain if oxygenation is possible. This technique may be useful in both predicted and unexpected difficult intubation and also in cases where neuromuscular blocking agents are either contraindicated or not required. The side effects of succinylcholine, and also those of non-depolarizing drugs, such as anaphylaxis are avoided.

Laryngoscopy and endotracheal intubation are mandatory for most patients undergoing general anaesthesia, which is invariably associated with certain cardiovascular changes such as tachycardia or bradycardia, rise in blood pressure and a wide variety of cardiac arrhythmias. These effects are deleterious in susceptible individuals culminating in perioperative myocardial ischemia, acute heart failure and cerebrovascular accidents. The cardiovascular response to laryngoscopy and endotracheal intubation has been recognized since 1951. The response following laryngoscopy and intubation peaks at 1-2 minutes and returns to normal within 5-10 minutes.

Though these sympatho adrenal responses are probably of little consequence in healthy individuals, it is hazardous to those patients with

hypertension, coronary heart disease, intra cranial pathology and hyper reactive airways.

Various systemic as well as topical agents were used to reduce these untoward hemodynamic responses due to laryngoscopy and intubation. The common strategies adopted are narcotics, vasodilators, beta blockers, calcium channel blockers, lidocaine and other sympatholytics.

After the emergence of shorter-acting opioids like remifentanil and alfentanil, these drugs were combined with propofol for successful tracheal intubation without muscle relaxants. These drugs are not yet available in many developing countries. Fentanyl is the opioid commonly available and being used in combination with propofol, lignocaine and midazolam for intubation without muscle relaxants.

ANATOMY OF LARYNX

It is a protective sphincter of the respiratory tract and it contains the vocal cords. It contains muscles of the larynx, cartilages, ligaments and membranes. It extends from C3 - C6 cervical vertebrae. Measurements of the larynx include:

TABLE.I

Parameters	Males	Females
Length	44 mm.	36 mm.
Transverse diameter	43 mm.	41 mm.
Antero-posterior diameter	36 mm.	26 mm.
Circumference	136 mm.	112 mm

RELATIONS

Anteriorly - covered by the fascia, platysma and skin

Posteriorly - pharynx, prevertebral muscles and cervical vertebrae

Superiorly - pharyngeal structures

Inferiorly - continues as trachea

Cartilages of the Larynx

There are nine cartilages in the larynx, three of which are paired and three are unpaired.

TABLE.II

Unpaired cartilages	Paired cartilages
Thyroid	Two Arytenoid
Cricoid	Two Corniculate
Epiglottis	Two Cuneiform

- **Epiglottis:** Leaf like structure, the lower part of the epiglottis is attached to the thyroid cartilage by the thyro-epiglottic ligament, and the upper broader is free to project superiorly. Its posterior free surface forms a bulge called the tubercle. Valleculae are the region between the medial and lateral glosso- epiglottic fold and it is the most common site of fish bone impaction.
- **Thyroid cartilage:** Sheild like shape , the lower part of its two laminae join together to form a prominence in the males called Adam's apple; it is less prominent in females. It is the largest laryngeal cartilage and inferiorly it articulates with cricoid cartilage.

- **Cricoid cartilage (hyaline): ‘**signet ring’ shaped and it is situated at the level of C6 vertebrae. The lateral border articulates with the thyroid cornua, and on its upper border with the arytenoid cartilages.
- **Arytenoid cartilages:** Pyramidal in shape, each with a lateral muscular process (for insertion of both crico-arytenoid muscles) and an anterior vocal process (for the posterior attachment of the vocal ligament).
- **Corniculate cartilages:** present on the apex of the arytenoid cartilage.
- **Cuneiform cartilages:** It is a flake of cartilage within the margin of the ary-epigloltic fold.

LIGAMENTS

There are four extrinsic and intrinsic ligaments in the larynx.

- **Thyrohyoid membrane**: It stretches between the upper border of the thyroid and the hyoid bone.
- **Hyo-epiglottic ligament:** It connects the epiglottis to the back of the body of the hyoid
- **Cricothyroid ligament**: Lies between the thyroid cartilage and the cricoid cartilage, it is the preferred site for cricothyrotomy.
- **Cricotracheal ligament**: which links the cricoid cartilage to the first tracheal ring.

MUSCLES OF THE LARYNX

Extrinsic:

- Sternothyroid - depresses the larynx
- Thyrohyoid - elevates the larynx
- Inferior constrictor - constrictors of the pharynx

Intrinsic:

- Posterior crico-arytenoid –abducts the cord by external rotation of the Arytenoids.
- Lateral crico-arytenoid –adducts the cord by internal rotation of the Arytenoids.
- Inter arytenoid – closes the posterior part of the glottis
- Thyro-arytenoid – relaxes the cords by shortening the cords.
- Vocalis – fine adjustment of vocal cord tension
- Cricothyroid – tensor of the vocal cords

BLOOD SUPPLY

1. **Arterial supply**

- Superior laryngeal artery is a branch of superior thyroid artery;

  It runs with the internal branch of the superior laryngeal nerve
- Inferior laryngeal artery is a branch of the inferior thyroid artery,

  It accompanies the recurrent laryngeal nerve.

2. **Venous drainage** through the corresponding superior and inferior thyroid veins

Nerve Supply of Larynx

Vague is the main nerve supply of the larynx, divided into superior laryngeal nerve and recurrent laryngeal nerve.

The Superior Laryngeal nerve arises from the inferior ganglion of Vagus. It is further divided in to external and internal branches. The external branch provides motor supply to the cricothyroid muscle while the internal branch divides into two main sensory and secretomotor branches.

The upper branch supplies the mucous membrane of lower part of phaynx, epiglottis, vallecula and vestibule of larynx. The lower branch supplies the aryepiglottic fold and mucous membrane down to the level of vocal folds.

The Internal branch of superior laryngeal nerve supplies the mucosa above the level of glottis.

The Recurrent laryngeal nerve ascends to the larynx in the groove between the oesophagus and trachea and divides into motor and sensory branches.

The motor branch supplies all the intrinsic muscles of larynx except the cricothyroid.

The sensory branch supplies the laryngeal mucous membrane below the level of vocal folds.

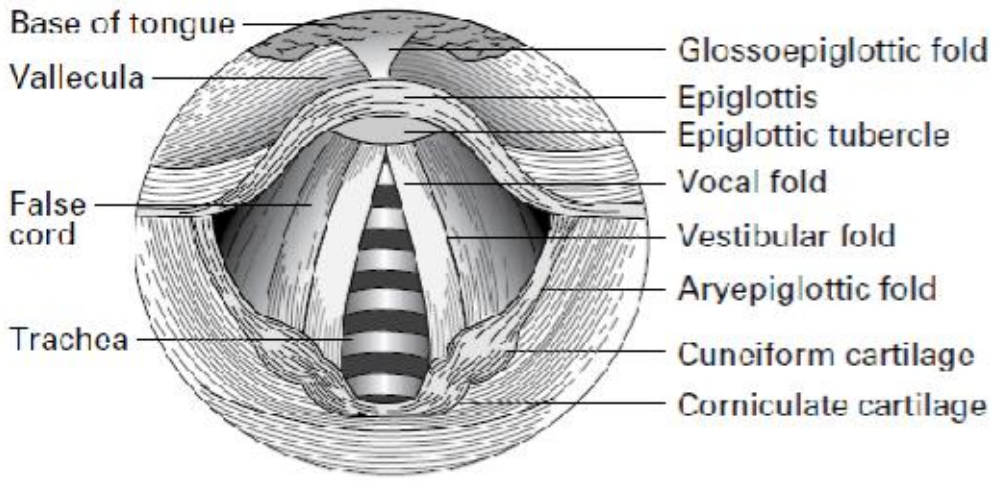
LARYNGOSCOPIC ANATOMY

Oral, pharyngeal and laryngeal axis should be in the same plane for laryngoscopy and intubation.

During direct laryngoscopy the following structures are seen in the order: the base of the tongue, the valleculae, the anterior surface of the epiglottis and the aryepiglottic folds containing cuneiform and corniculate cartilages.

The vocal cords appear as pale, glistening structure that extends from the angle of the thyroid cartilage to the vocal processes of the arytenoids. Between the vocal cords the triangular opening is called rimaglottidis, through which the upper two or three tracheal rings can be visualized.

LARYNGOSCOPIC ANATOMY



LARYNGEAL AXIS

In supine position the oral, pharyngeal and laryngeal axes of the patient are offset, making it difficult to obtain a good view of the glottis by the conventional laryngoscope. Flexion of neck (25 to 35 degree) causes alignment of pharyngeal and laryngeal axis in the same plane. Then subsequent head extension at the atlanto occipital joint (80 to 85 degree) causes alignment of oral axis with the pharyngeal and subsequently with the laryngeal axis. This position (neck flexion and head extension) is called optimal sniffing position.

In adults, a head elevation of 8 – 10 cm, as on a pillow or doughnut, achieves appropriate neck flexion. No such head elevation is required in pediatric age group as their large head circumference size produces neck flexion as the head is extended at atlanto-occipital joint.

The sniffing position has been recommended as the optimal one for intubation and airway management. Historically, the definition of this position is credited to an Irish born anaesthetist, Sir Ivan Magill (1936), who described it as “sniffing the morning air” or “draining a pint of beer”.

Banister and Macbeth described the technique and they analysed the angles of the oral, pharyngeal, and laryngeal axes with the head in different positions for the purpose of identifying the best possible alignment of the three axes to expose the glottis and facilitate endotracheal tube insertion.

The key components are flexion of the lower cervical spine, extension of the upper cervical spine and atlanto-occipital joint.

The main advantage of this position is the optimal exposure of the glottis for the purpose of intubation. The disadvantage include its inadequacy in obese patients to optimize glottis exposure by direct laryngoscopy. It is contraindicated in patients with known or suspected cervical injuries.

MALLAMPATTI CLASSIFICATION

This is probably the most commonly employed test for predicting difficult airway. It indicates the amount of space within the oral cavity to accommodate the laryngoscope and ETT. Performing the test meticulously is critical to correct prediction. This is performed by having the patient open the mouth as wide as possible and stick out the tongue without phonation. One should also ensure that the patient is in the sitting position with the head protruding forward, mimicking the “sniffing” position of laryngoscopy and intubation. The observer’s eye should be at level of the patient’s open mouth so that the faucial pillars, uvula, soft palate and the hard palate are visible. As per Samsoon and Young’s modification of Mallampati grading, following 4 grades may be noted :

Grade I : Faucial pillars, uvula, soft and hard palate visible.

Grade II : Uvula, soft and hard palate visible.

Grade III : Base of uvula or none, soft and hard palate visible.

Garde IV : Only hard palate visible.

Grade I and II are associated with easy laryngoscopic view of the glottis.Grade III and IV implies difficult viewing of the glottis by conventional laryngoscopy.

CORMACK AND LEHANE GRADING OF LARYNGOSCOPIC VIEW

Grade I : Visualization of entire vocal cords.

Grade II : Visualization of posterior part of vocal cord.

Grade III : Vsualization of epiglottis.

Grade IV : No glottic structures seen.

Cook (1999) has further subdivided Cormack and Lehane's Grade II and III into IIa, IIb, IIIa, and IIIb. II a and II b indicates visualization of posterior part of vocal cord and tip of the arytenoids respectively.III a indicates liftable epiglottis and III b indicates adherent epiglottis. As per Cook, grade I and IIa patient can be directly intubated, IIb and IIIa would require bougie while IIIb and IV cannot be intubated using conventional laryngoscope and bougie, but would require alternative specialized techniques.

PATHOPHYSIOLOGY OF LARYNGOSCOPY AND INTUBATION RESPONSE

PATHOPHYSIOLOGY OF CARDIOVASCULAR CHANGES DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

Tracheal intubation alters the respiratory and cardiovascular physiology by both reflex response and by physical presence of endotracheal tube. Although reflex responses are generally of short duration and of little consequence in majority of patients, they may produce profound disturbances in patients with underlying abnormalities such as hypertension, coronary heart disease, reactive airways, and intracranial pathology.

CARDIOVASCULAR RESPONSE

The common cardiovascular responses to laryngoscopy and endotracheal intubation are increased blood pressure and heart rate, mediated by the cardio accelerator nerves (sympathetic efferent) and sympathetic chain ganglia. This autonomic response following endotracheal intubation is due to release of norepinephrine from adrenergic nerve terminals and adrenal medulla.

Increased blood pressure following intubation results from the activation of renin angiotensin system. Its activation releases the renin from the renal juxta glomerular apparatus of the kidney innervated by beta adrenergic nerve terminals.

The effects of endotracheal intubation on the pulmonary vasculature are less well understood than the responses elicited in the systemic circulation. They are often coupled with the changes in airway reactivity associated with intubation. They are i) glottis closure reflex (laryngospasm due to brisk motor response, ii) decrease in dead space, iii) increase in airway resistance, iv) bronchospasm (a reflex response to intubation), v) removal of glottis barrier and reduction in lung volume, vi) reduction of efficiency of cough reflex.

Methods used to decrease cardiovascular responses to laryngoscopy and intubation.

To reduce the risk of peri operative ischemia and infarction the balance between the myocardial oxygen supply and demand should be maintained.

Factors affecting myocardial oxygen demand and supply:

Demand

- Basal requirement
- Heart rate
- Wall tension-preload, after load
- Contractility

Supply

- Heart rate – depends on diastolic time. Increases in heart rate shorten diastolic time, resulting in decreased oxygen supply to myocardium.
- Coronary Perfusion Pressure –CPP increases with high aortic diastolic pressure and low ventricular end diastolic pressure.
- Arterial oxygen content – depends on arterial oxygen partial pressure and haemoglobin concentration.
- Coronary vessel diameter.

Increasing depth of General Anaesthesia

Inhalational agents are used to blunt the cardiovascular responses during laryngoscopy and endotracheal intubation. This is achieved by increasing the concentration of inhalational agents resulting in profound cardiovascular depression prior to laryngoscopy and intubation. Various agents used are Halothane, Isoflurane, and Sevoflurane.

1. Lidocaine

- Lidocaine gargle for oropharyngeal anaesthesia.
- Aerosol for intra-tracheal anaesthesia
- Topical spray for vocal cords
- Regional nerve blocks - Superior Laryngeal nerve, Glossopharyngeal nerve
- Intra venous bolus of systemic anaesthesia.

Mechanism of action of Intravenous lignocaine

- By Increasing the depth of General Anaesthesia
- Potentiation of effects of nitrous Oxide and reduction of MAC of Halothane by 10-28%
- Direct Cardiac depressant
- Peripheral Vasodilation
- Antiarrhythmic properties
- Suppression of cough reflex.

2. Vasodilators

- Hydralazine : Bolus: 5-20 mg, infusion 0.25 -1.5 µg/kg/min
- Sodium nitroprusside: Bolus:50-100 µg, infusion 0.5-10 µg/kg/min
- Nitroglycerine: Bolus: 50-100 µg, infusion 0.5-10 µg/kg/min

3. Narcotics

- Fentanyl - 2µg/kg,
- Alfentanil - 20µg/kg,
- Remifentanil - 2µg/kg

Mechanism of action of opioids

- Suppression of Nociceptive Stimulation caused by Intubation
- Centrally mediated decrease in sympathetic tone
- Activation of vagal tone.

4. Adrenergic blockers

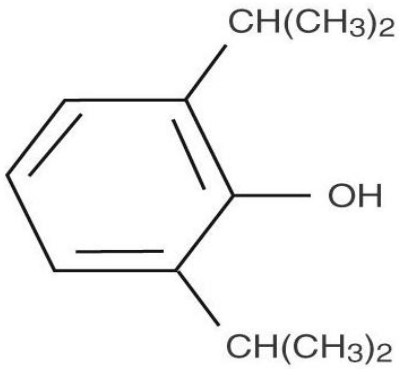
- Long acting - Metoprolol 1- 15 mg,
  Propronalol 1- 10 mg.
- Short acting - Esmolol 0.5mg/kg followed by 50-200µg/kg/min.

5. Alpha$_2$ agonist – Clonidine.
6. Midazolam – Sedative & Anxiolytic
7. MagnesiumSulphate – Sedative & Anxiolytic.

PHARMACOLOGY OF PROPOFOL[21]

CHEMICAL STRUCTURE

Chemical name is 2,6-di-isopropylphenol



COMMERCIAL PREPARATION

It is a white oil-in-water emulsion, it contains

1% - propofol

10% - soyabean oil

2.25% - glycerol

1.25% - purified egg phosphatide with a pH -7.

MECHANISM OF ACTION

It is a selective modulator of GABA–A receptor, acts by increasing the transmembrane Cl^- conductance resulting in hyperpolarization.

It also reduces the dissociation of GABA from its receptors leading, to increase in the duration of Cl^- channel opening.

PHARMACOKINETICS

Volume of distribution (L/kg)	:	3.5 – 4.5
Context sensitive $t^{1/2}$(min)	:	40
Elimination $t^{1/2}$(hrs)	:	0.5 -1.5
Clearance (ml/kg/min)	:	30-60

CLINICAL USES

Propofol is the induction agent of choice, where conditions required rapid and complete awakening after anaesthesia.

INDUCTION OF ANAESTHESIA

Dose: 1.5-2.5mg/kg IV Higher dose required in children. Elderly patients require lower doses. Complete awakening without residual CNS effects is the characteristic feature of Propofol.

INTRAVENOUS SEDATION

Dose : 25-100 µg/kg/min, Fast recovery without residual effect. Used as a sedative during mechanical ventilation in ICU.

Provides control of stress responses and has anticonvulsant and amnestic properties.

MAINTENANCE OF ANAESTHESIA

Dose : 100-300 µg/kg/min, in combination with opioids or midazolam used in short ambulatory procedures. Minimal postoperative nausea and vomiting.

NON HYPNOTIC THERAPEUTIC APPLICATIONS

ANTI EMETIC EFFECTS

Dose : 10-15mg IV, sub hypnotic dose. Used in post anaesthesia care unit to treat nausea and vomiting, chemotherapy induced nausea and vomiting.

ANTI PRURITIC EFFECTS

Dose : 10 mg IV, Used in treatment of pruritus associated with opioids and cholestasis.

ANTI CONVULSANT ACTIVITY

Acts by GABA-mediated presynaptic and post synaptic inhibiton of cl^- channels.

ATTENUATION OF BRONCHOCONSTRICTION

Reduces the incidence of wheezing after induction and endotracheal intubation both in healthy and asthmatic patients.

EFFECTS ON ORGAN SYSTEMS

CENTRAL NERVOUS SYSTEM

It decreases the cerebral metabolic rate for oxygen (CMRO2), cerebral blood flow and intracranial pressure (ICP).Cerebral autoregulation and cerebral blood flow to changes to Paco2 are not affected. Higher doses produce burst suppression in EEG.

CARDIOVASCULAR SYSTEM

It reduces the blood pressure and heart rate. These effects are increased in elderly, coronary heart disease and hypovolemic patients. Heart rate responses to intravenous atropine are attenuated in patients receiving propofol, due to suppression of sympathetic nervous system activity. Treatment for propofol induced bradycardia is β-agonist Isoproterenol.

LUNGS

Produces dose dependent depression of ventilation in 25%-35% patients. Preoperative medication like opioids may enhance this ventilatory depressant effects. Produces bronchodilation and decreases the incidence of intra operative wheezing in asthmatic patients.

HEPATIC AND RENAL FUNCTION

No significant adverse effects.

INTRAOCULAR PRESSURE

Decreases intraocular pressure after induction and intubation.

SIDE EFFECTS

Allergic Reactions

Allergic components include phenyl nucleus and diisopropyl side chain.

Lactic Acidosis

Also known as propofol infusion syndrome. Occurs in patients receiving high dose infusions 75µg/kg/min for 24hrs. Unexplained tachycardia during anaesthesia should raise the suspicion of metabolic acidosis. Laboratory evaluation includes arterial blood gases and serum lactate. It is reversible in early stage with the discontinuation of drug.

PROCONVULSANT ACTIVITY

Due to spontaneous excitatory movements of subcortical origin. Prolonged myoclonus associated with meningismus.

BACTERIAL GROWTH

It supports the growth of E.coli and Psedomonas aeruginosa. Aseptic technique should be used while handling. Contents should be used within 6hrs after opening of the vial.

INJECTION PAIN

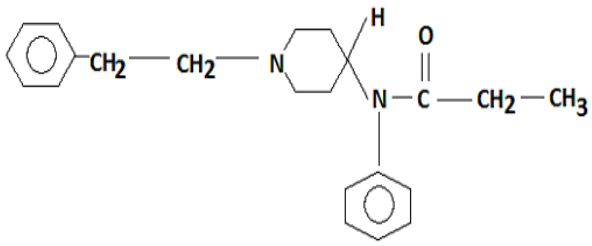
Injection pain is reduced by prior administration of 1% lignocaine or a short acting opioid.

ANTI OXIDANT PROPERTIES

Similar to vitamin E

PHARMACOLOGY OF FENTANYL[22]

CHEMICAL STRUCTURE



Fentanyl is synthetic phenylpiperidine opioid of the 4-anilopiperidine series which is structurally related to pethidine.

COMMERCIAL PREPARATION

Commercially fentanyl is formulated as a citrate, available as an aqueous solution without preservatives. Each ml contains a base of 50µg of fentanyl citrate.

PHARMACOKINETIC PROFILE

Molecular Weight	: 528.29
Pka	: 8.4
Unionized form in pH 7.4	: 8.5
Octanol / water partition coefficient	: 816
Bound to plasma proteins (Percentage)	: 84
Potency	: 80 times more potent than morphine

Pharmacodynamics

Analgesia

This results from action of fentanyl on opioid μ receptors both supra spinally in the brain and in the spinal cord. Intravenous fentanyl produces effective analgesia at plasma concentrations between 0.6-3.0ng/ml.

Cardiovascular system

Arterial blood pressure, cardiac output and pulmonary vascular resistance remain unchanged after large doses of intravenous fentanyl. Fentanyl like other opioid agonists (expect pethidine) causes bradycardia that responds to intravenous atropine. Peripheral vasodilation is much less than morphine due to absence of histamine release.

Respiratory System

Fentanyl causes a direct dose related respiratory depression by its depressant effect on the medullary respiratory center, manifested as a decreased sensitivity to carbondioxide and reduced respiratory rate. It is reversed by intravenous Naloxone administration. Fentanyl concentrations in the plasma >2ng/ml is associated with respiratory depression. The respiratory depression depends on various factors, including type of the surgical procedure, age of the patient and individual pharmacodynamic response.

Central nervous System

Fentanyl causes less sedation than equianalgesic doses of morphine. In doses of 100 μg, fentanyl causes dose related reduction in cerebral blood flow and CMRO2. Catatonic state due to fentanyl injection is manifestated as muscle rigidity, due to increased dopamine biosynthesis in the caudate nucleus.

Gastrointestinal system

Fentanyl decreases gastrointestinal tract motility, increases intra gastric pressure and causes a varying incidence of nausea and vomiting. It is due to chemoreceptor trigger zone stimulation in the area postrema.

Genito-urinary System

Fentanyl like other opioids causes relaxation of detrusor muscle and increase in urethral sphincter tone leading to urinary retention. This is probably not dose related and is more common with central neuraxial administration.

Pharmacokinetics

Fentanyl is a potent opioid, highly lipophilic, producing a rapid onset of action of relatively short duration. After intravenous administration, it is fastly distributed to Heart, Brain and highly perfused tissues. It crosses the placental barrier. Peak effect occurs in 5 minutes. Within a short time, the drug redistributes to inactive tissue sites like skeletal muscle and fat, associated with decrease in plasma concentration of drug, thus terminating its effect. About 75% of initial dose undergoes first pass pulmonary uptake.

When low doses (1-2µg/kg) are administrated, redistribution terminates the effect and the drug appears short acting. With administration of large intravenous doses or continous infusion, progressive saturation of inactive tissue sites occur, with redistribution becoming insufficient to terminate drug action which becomes dependent on slow elimination process and the drug appears to be long acting .

Pharmacokinetic profile

Volume of distribution of steady state	: 335litres
Clearance	: 539 ml/min
Effect-site equilibration time	: 6.8min
Hepatic extraction ratio	: 0.8-0.1
Context –Sensitive t ½ (4 hrs infusion)	: 260 min
Elimination t ½	: 3.1 to 6.6 hours.

Metabolism

Fentanyl is biotransformed in the liver to inactive metabolites, primarily norfentanyl and several hydroxylation products. Only 4-7 % of drug is excreted unchanged in urine. Elimination t ½ of fentanyl is longer than that of morphine because of high lipid solubility of fentanyl. Elimination t ½ is prolonged in elderly patients. A high hepatic extraction ratio means that the clearance of fentanyl is limited by hepatic blood flow.

Routes of Administration and Dosage

Intramuscular

50 -100μg may be administrated intramuscularly as premedication 30 to 60 minutes prior to surgery.

Intravenous

Can be given intra operatively and for postoperative analgesia. Postoperative pain relief is given by intravenous bolus dose of 1-2μg/kg followed by an infusion dose of 1-2μg/kg/hr. In Patient Controlled Analgesia (PCA) bolus dose is 20-50μg with lockout intervals.

Transdermal

Transdermal fentanyl patch is available in four sizes; it provides sustained release of fentanyl citrate at rates of 25 μg/hr, 50 μg/hr, 75μg/hr and 100μg/hr over a period of 48-72 hrs. Skin acts as a secondary reservoir contributing to prolonged residual fentanyl concentrations.

Transmucosal

Oral transmucosal fentanyl citrate contains fentanyl citrate in a candy shaped into a stick. Time to onset of analgesia is 4minutes and the duration of analgesia is 150minutes.

Intranasal

It is also administrated with a metered dose device. During each spray it delivers 4.5μg fentanyl. Time to onset of analgesia is about 15 minutes.

Transpulmonary

Inhalational route of fentanyl administration produces rapid, effective drug delivery. A dose of 300μg of fentanyl administrated via oxygen driven nebulizer produces effective postoperative analgesia in 5 min and lasts for about 2 hours.

Clinical Application

Premedication

Fentanyl in doses of 50-100μg may be administrated intramuscularly 30-60 minutes prior to surgery. Oral transmucoal fentanyl citrate in doses between 15-20μg/kg, administrated 45 minutes before surgery produces reliable preoperative sedation and facilities induction of anaesthesia in children.

Adjunct to general anaesthesia

Fentanyl in doses of 1-2μg given intravenously provides analgesia. It is used as an adjuvant to decreases the cardiovascular responses that occur during direct laryngoscopy for intubation and surgical stimulation. Large doses of fentanyl, 50-150μg/kg intravenously has been used as sole anesthetic agent especially in cardiothoracic procedures, principally because of its stable hemodynamic effects.

Neruolept analgesia

It is a premixed combination, containing 2.5mg Droperidol and 0.05mg Fentanyl in each ml (50:1) used for neuroplept analgesia and anaesthesia.

Adjunct in Central neuraxial Block

Fentanyl added to local anesthetic either intrathecally or epidurally, improves the quality of intraoperative analgesia and also provides good post operative analgesia.

Postoperative analgesia

Fentanyl administration by intravenous, epidural, intrathecal and transdermal routes provides effective postoperative analgesia. Newer routes like intranasal and inhalational administration are being evaluated as minimally invasive means of postoperative analgesia.

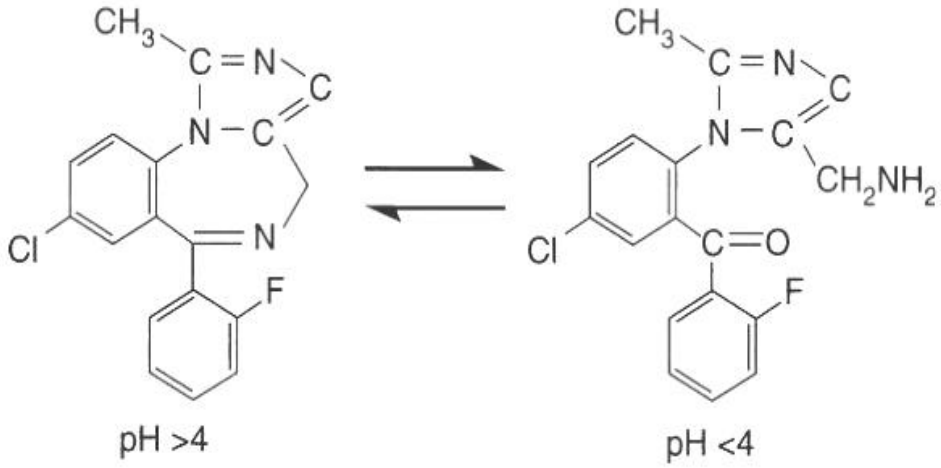
Side effects

Commonly occurring side effects include dose dependent respiratory depression, nausea and vomiting, pruritus, urinary retention and bradycardia. These effects are reversed by administration of Naloxone intravenously.

PHARMACOLOGY OF MIDAZOLAM[23]

CHEMICAL STRUCTURE

Midazolam belongs to Benzodiazepine group. The imidazole ring in the structure of the midazolam is responsible for its stability in aqueous solutions and its rapid metabolism. Amnestic effects of midazolam is more potent than its sedative properties. It is 2-3times more potent than diazepam. After its administration patient may be awake but remain amnestic for events and conversations for several hours.



COMMERCIAL PREPARATION

Available as an aqueous solution with solubilizing preparation like propylene glycol. It is compatible with acidic salts of drugs like opioids, anticholinergics and Ringer lactate.

PHARMACOKINETICS

TABLE.III

Volume of distribution (L/kg)	1-1.5
Protein Binding (%)	96-98
Clearance (ml/kg/min)	6-8
Elimination $t^{1/2}$(hrs)	1-4
Effect site equilibration time (min)	0.9-5.6

METABOLISM

It is metabolized by hepatic and small intestinal enzymes CYP450 (CYP3A4). The metabolism is slowed by CYP450 inhibitors like cimetidine, erythromycin, calcium channel blockers and antifungals. Its active metabolite is 1-OH midazolam.

RENAL CLEARANCE

Pharmacokinetic property is not altered by renal failure.

EFFECTS ON ORGAN SYSTEMS

CENTRAL NERVOUS SYSTEM

It reduces the cerebral metabolic oxygen requirement (CMRO2) and cerebral blood flow. It is a potent anticonvulsant, effective in the treatment of status epilepticus. It does not possess neuroprotective activity.

RESPIRATORY SYSTEM

Depresses the ventilation, mostly pronounced effect seen in COPD patients. Produces transient apnoea after rapid administration of large doses. It also depresses the swallowing reflex and upper airway activity.

CARDIOVASCULAR SYSTEM

Decreases blood pressure as like other induction agents, more pronounced in hypovolemia patients.

CLINICAL USES

PREOPERATIVE MEDICATION

Most commonly used oral preoperative drug for children. It is effective for producing sedation and anxiolysis.

Dose: 0.25mg/kg

INTRAVENOUS SEDATION

Dose	:	1-2.5mg i.v
Onset	:	30-60sec
Time to peak effect	:	3-5min
Duration of sedation	:	15-80min

Exaggerated response on ventilation in the presence of opioids and other CNS depressant drugs. Increasing age greatly increases the pharmacodynamic sensitivity to the hypnotic effects.

INDUCTION OF ANAESTHESIA

Dose: 0.1-0.2 mg/kg IV over 30-60sec. Onset of unconsciousness is facilitated by the preceding injection of small dose of opioid. Exaggerated cardiovascular responses seen in the presence of other CNS depressant drugs like propofol and thiopentone.

MAINTENANCE OF ANAESTHESIA

Supplemental effect with opioids and propofol. Produces Dose-dependent decrease in the requirement of volatile anaesthetics. .

POSTOPERATIVE SEDATION

Loading dose : 0.5 - 4mg IV

Maintanence dose : 1-7mg/hr IV

Emergence time is increased in elderly, obese and severe liver disease patients.

SIDE EFFECTS

Physical and psycological dependence with withdrawal symptoms. Oversedation and seizure like activity (paediatrics), Euphoria, Reduced alertness, confusion, hallucinations, dizziness, ataxia and anterograde amnesia, Skin rash, pruritus and Anaphylaxis.

Fatal complications include

Respiratory depression and respiratory arrest, Hypotension.

PRECAUTIONS

Dose should be reduced in elderly and debilitated patients.

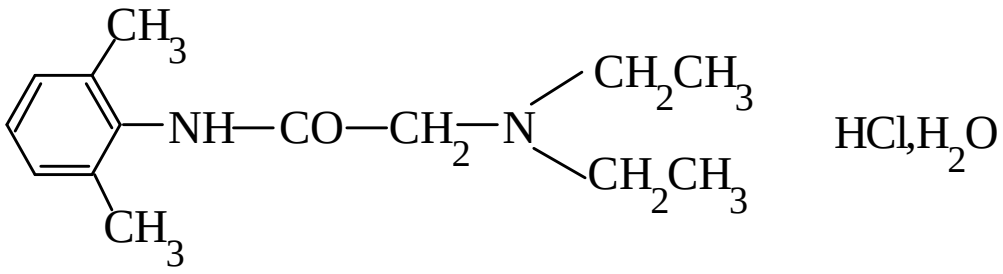
OVERDOSAGE

Sedation, confusion, impaired coordination, muscle relaxation. Areflexia, hypotension, cardio-respiratory depression, apnoea and coma.

Treatment is supportive and symptomatic. In severe intoxication benzodiazepine antagonist Flumazenil is indicated.

PHARMACOLOGY OF LIGNOCAINE[24]

Lignocaine is local anaesthetic, which belongs to the amide group of anaesthetic agents. It is used in wide range of anaesthetic applications. It is also useful in other systemic diseases such as cardiac arrhythmias associated with myocardial infarction and in status epilepticus. Adult patients with normal cardiac output, hepatic function, and hepatic blood flow, an initial bolus dose of lidocaine 1.5- 2 mg/kg, followed by a infusion dose of 1 to 4 mg/min should provide therapeutic plasma lidocaine concentrations of 1 to 5 µg/ml.



Physical properties

TABLE.IV

Relative potency	1
Pka	7.9
Plasma protein binding	70%
Structure	Amide
Stability	+++
Molecular weight	234
Adrenaline compatibility	+++
Maximum dosage	3mg/kg, (with adrenaline 7 mg/kg)
Lipid solubility	2.9
Elimination half time	96 min
Clearance	0.95 l/min
Toxic plasma concentration	More then5µg/ml

Pharmacokinetics

Lignocaine is readily absorbed from the mucous membranes and damaged skin and also from the injection sites (muscle).

After an intravenous dose, lignocaine is fastly distributed into highly perfused and vascular tissues followed by redistribution into the skeletal muscle and adipose tissues. About 60-80% of the lignocaine is bound to alpha-1 acid glycoprotein and it depends on the concentration of the drug used.

The plasma concentrations of the lignocaine fastly decrease after an intravenous dose with a $t_{1/2}$ of less than 30 minutes. When infusions are given for a longer time, the elimination half-time may be prolonged for longer than 24 hours.

Lidocaine is highly metabolized in the liver and alterations in the hepatic blood flow can affect the pharmacokinetics of the drug. Reduced clearance of this agent is found in patients with congested liver and in alcoholic liver disease.

The active metabolite is mono-ethylglycine xylidide, which may also accumulate in patients with reduced cardiac output.

Metabolism of lignocaine is not affected by renal impairment but the accumulation of its active metabolite can lead to toxicity. The progressive increase in the concentration of AAG (alpha-1 acid glycoprotein) as seen after lignocaine during its infusion in these patients.

Surface application of lignocaine gives rise to slow rise in serum concentrations. Acceptable plasma lignocaine concentrations have been produced by local application jelly in certain procedures such as bronchoscopy.

Uses of lignocaine

TABLE.V

Clinical use	Concentration (%)	onset	Duration (min)	Recommended maximum single dose(mg)
Topical	4 %	fast	30 – 60	300
Infiltration	0.5 – 1 %	fast	60 – 240	500 with epinephrine
IVRA	0.25 – 0.5 %	fast	30 – 60	300
Peripheral nerve block	1 – 1.5 %	fast	60 – 180	500 with epinephrine
Epidural	1.5 – 2 %	fast	60 – 120	500 with epinephrine
Spinal	1.5 – 5 %	fast	30 – 60	100

Lignocaine is a local anaesthetic widely used by injection and for local application to mucous membranes. The speed of onset and absorption into the circulation depend upon the addition of vasoconstrictors during application.

The dosage required in peripheral nerve blocks depends upon the route of administration and the site of administration. It is used along with adrenaline to increase its efficacy and the time of action. As a 1% solution, it is used for sympathetic nerve block in doses of 5 ml for cervical and in 10 ml doses in lumbar block.

In epidural anesthesia, 2 to 3 ml solution is needed for each dermatome, but the total doses recommended are not to exceed 30 ml and a maximum of 2% concentration.

For continuous epidural anesthesia, the maximum doses should not be repeated more than once in 90 minutes. A hyperbaric solution of 1.5% lignocaine in 7.5% glucose has been used for spinal anaesthesia. Doses up to 1 ml of solution have been used for obstetric surgeries.

For intravenous regional anesthesia, a 0.5% solution has been used in up to 50 ml.

Its use as an anti-arrhythmic agent has been proved for many decades. Its specific use is in the treatment of ventricular tachyarrhythmias. It is also the drug of choice for ventricular arrhythmias associated with acute myocardial infarction and is used in cardiopulmonary resuscitation and ventricular fibrillation, when there is no response to cardioversion. In cardiopulmonary resuscitation, it is given

as a single dose of 100 mg. In other conditions, it is given as a bolus dose followed by an infusion of 1 to 1.5 mg/kg body weight as a direct intravenous injection at the rate of 25 mg/min. If no effect is seen in 5 minutes, the loading dose may be repeated to a maximum dose to a maximum dose of 2000 to 3000 mg in 1 hour. It is rarely necessary to continue the infusion for longer than 24 hours.

Epilepsy is another indication in situations such as status epilepticus when diazepam and phenytoin are ineffective. In adults, 100 mg may be given by slow intravenous injection followed by an infusion at the rate or 1 to 2 mg per minute. Occasionally, recurrence of seizures may be seen on withdrawal of prolonged lignocaine therapy.

Lignocaine has been used to attenuate the pressor response induced by intubation.

In certain pain syndromes such as diabetic neuropathy and in other chronic painful disorders, lignocaine has proved to be useful. Other painful conditions such as perineal trauma as in episiotomy benefit with local spray of 5% lignocaine. Spinal anesthesia and epidural anaesthesia are the main and important uses of lignocaine.

ADVERSE EFFECTS

Central Nervous System

Reports of suspected psychotic reactions associated with lignocaine have been published in 6 patients given lignocaine for cardiac arrhythmias.

Cardio Vascular System

Lidocaine is essentially devoid of effects on the ECG or cardiovascular system when the plasma concentration remains <5 µg/ml cardiovascular side effects at therapeutic cocentrations are rare, except in patients with pre-existing compromised ventricular function. Conduction disturbances are rare. High plasma lidocaine concentrations may lead to arrhythmias, hypotension, heart block, and cardiac- respiratory arrest.

Allergic Reaction

Allergic reaction following lignocaine includes itching, skin rash, swelling of skin, Difficulty in breathing.

Clinical effects of overdose

TABLE.VI

Plasma lidocaine concentration (µg/ml)	Effect
1 – 5	Analgesia
5 – 10	Circumoral numbness,Tinnitus, Skeletalmuscle twitching, Systemic hypotension, myocardial depression
10 – 15	Seizures Unconciousness
15 – 25	Apnea Coma
More than 25	Cardiovascular depression

TREATMENT FOR OVERDOSE

Severe reactions

Immediately stop the drug administration; monitor the vitals. Airway maintenance with 100% administration of oxygen. **Circulatory depression**: vasopressors and intravenous fluids.

Seizures: Diazepam in 2-5mg increments, or an ultra-short-acting barbiturate (such as thiopental or thiamylal) in 50 to l00 mg increments, is given.

Skin: Fixed drug eruptions have been described.

Precautions

An intradermal test dose is always safe before lignocaine is used for any injectable anaesthesia. It should not be used in patients with hypovolemia and heart block or by other conduction disturbances. In patients with cerebrovascular disorders, lignocaine should be used with caution, as it may reduce cerebral blood flow.

Its use with other drugs such as beta blocking agents is rather unsafe, as the concentration of lignocaine in the plasma may be increased. Its use with cimetidine has been extensively studied.This drug reduces the lignocaine clearance from the plasma. Ranitidine has not been shown to have any effect on lignocaine kinetics. Lignocaine should not be used to treat arrhythmias induced by cocaine intoxication.

Lignocaine is not safe in patients with porphyria, because in animal studies it has been shown to be a porphyrinogenic substance. Smokers have been shown to have a reduced systemic bioavailability of lignocaine.

Antiarrhythmic

loading dose	:	2% Lignocaine 1.5 mg/kg (without perservative)
Infusion doae	:	1 to 4 mg/min (20 to 50 µg/kg/min)

REVIEW OF LITERATURE

Lewis CB et al [15](1948) used thiopentone sodium for tracheal intubation without using neuromuscular blocking agents. In 200 patients, either a blind nasal or direct oral intubation was done after thiopental 500–750 mg intravenously. There were two failures in the blind nasal group and six in the direct laryngoscopy group. Lewis encountered problems like coughing, laryngospasm. He demonstrated that adequate conditions for intubation could be achieved using thiopentone sodium alone.

Himes et al[8] (1977) studied the interactions between lidocaine and the anesthetics N_2O and halothane and confirmed that lidocaine reduced the dose of N_2O and Halothane for achieving the depth of anaesthesia in intraoperative period.

Poulton et al [18] (1979) compared the effectiveness of intravenous lidocaine and bronchodilator inhalation treatment in patients with chronic obstructive pulmonary disease (COPD) for rapid suppression of cough. Both lidocaine and bronchodilator inhalation treatments were equally effective for rapid cough suppression in patients with COPD.

Cormack RS, Lehane et al[4] (1984) studied difficult tracheal intubation in obstetric patients. They documented that the laryngeal view during direct laryngoscopy showed a fair inter observer reliability and

poor intra observer reliability, even when done by physicians well familiar with this rating system under standardized conditions.

Yukiola et al[32] (1985) studied the cough suppressant effect of various doses of intravenous lidocaine during tracheal intubation**.** He found that significant suppression of cough was achieved when 2 mg/kg of lidocaine was injected intravenously, between 1 and 5 min before attempting intubation. Cough reflex was suppressed completely when plasma concentrations of lidocaine exceeds 3 µg/ml.

Keaveny et al[13] (1988) showed the results of using two different doses of propofol 2.5 and 3 mg/kg, lignocaine 1.5 mg/kg, fentanyl 2µg /kg for assessing the intubating conditions. Clinically acceptable intubating conditions were obtained in 96.7% of patients in group receiving 2.5 mg/kg propofol compared to 100% in group receiving 3 mg/kg propofol without significant hemodynamic changes and 100% success can be obtained with 3 mg kg of propofol .

Ben Shlom et al[1] (1990) studied and found that Midazolam acts in synergism with fentanyl for inducing anaesthesia. 25% of the ED_{50} of fentanyl was required in combination with 23% of the ED_{50} for midazolam to achieve the ED_{50} of the combination. Midazolam was found to act in synergism with fentanyl for induction of anaesthesia. This synergistic action is due to mutual potentiation between opioids and benzodiazepines receptors.

Saarnivaara et al[26] (1991**)** studied the intubating conditions and cardiovascular changes following administration of propofol alone or in combination with alfentanil. He reported that only 5 out of 13 patients (38%) had proper intubating conditions. He found good to moderate intubating conditions in 79% of patients receiving a combination of propofol 2.5 mg/kg with alfentanil 30 µg/kg.

Short et al[27] (1991) studied the synergistic interactions between propofol and midazolam for induction of anaesthesia. The exact mechanism for this action is not known, but hypothesised to be an interaction at CNS GABA -A receptors. Propofol dose requirement was reduced to 52% in the presence of midazolam.

Mulholland et al[17] (1991) studied intubation with propofol augmented with intravenous lidocaine. He found that propofol 2.5 mg/kg suppressed the laryngeal reflexes which was augmented by the cough suppressive effects of lignocaine1.5 mg/kg.

Hookah et al[9] (1991) showed the induction of anesthesia with protocol in combination with lignocaine 1.5 mg/kg. This study compared the ease of performing laryngoscopy and endotracheal intubation without using muscle relaxants after the induction of anaesthesia with either propofol or thiopentone in 106 patients posted for elective surgery. Thiopentone sodium (5 mg/kg) or propofol (2.5 mg/kg), augumented with lidocaine (1.5 mg/kg) and alfentanil (30 µg/kg), were used. Jaw relaxation, visualisation of the larynx, vocal cords position, ease of

intubation and the tracheal tube tolerance are assessed. Jaw relaxation and open vocal cords were found in most patients in both groups. Visualisation of the larynx was good in 60 patients (46%) and intubation was easy in 48 (22%) of the patients given thiopentone and propofol, respectively.

Barker et al[2] (1992) studied the difference in laryngeal reflexes on intubation with propofol and thiopentone sodium. It showed suppression of laryngeal reflexes by propofol and this may account for the lower incidence of laryngospasm after induction of anaesthesia with propofol in comparison with thiopentone.

Davidson et al[5] (1993) showed the effect of intravenous lidocaine on the tracheal intubation after induction of anaesthesia with propofol and alfentanil. He found intubating conditions were better and there was less coughing when lignocaine was given before propofol and alfentanil

Grange et al[6] (1993) showed the effect of lignocaine or alfentanil with propofol for tracheal intubation without the use of muscle relaxants. Forty five patients posted for elective surgery were randomly allocated to receive either 0.9% saline group (control), alfentanil 20µg/kg group, or lignocaine 1.5 mg /kg group prior to induction with propofol 2.5 mg/ kg and to assess the ease of intubation. Alfentanil group shows better intubating conditions in 93% of patients compared to 60% in each of the groups pre-treated with lignocaine. He showed pretreatment with lignocaine was no better than saline.

Hiller et al[8] (1993) studied in children, the tracheal intubation after induction of anaesthesia with propofol, three different doses of alfentanil and lidocaine without using neuromuscular blockers. His studies showed that the best intubating conditions in children were produced by propofol 3.5 mg/kg and alfentanil40 µg/kg.

Kazama et al[14] (1997) studied the reduction of the CP_{50} values of propofol by using fentanyl and the hemodynamic responses to various noxious stimuli. He concluded that combined usage of Propofol and fentanyl suppressed motor and hemodynamic reactions to various noxious stimuli.

Grant et al[7] (1998) assessed intubating conditions in three groups of 60 patients, at 3 different doses of remifentanil. The patients were premedicated with temazepam and anaesthesia was induced with propofol 2 mg/kg and remifentanil 0.5, 1.0, or 2 µg/kg. Overall intubating conditions were regarded as acceptable in 20%, 50% and 80% of patients respectively. Remifentanil 2 µg/kg and propofol 2 mg/kg produced the best intubating conditions.

Stevens et al[28] (1998) studied tracheal intubation by remifentanil and propofol without muscle relaxants on ambulatory surgical cases.

Klemola et al[15] (2000) studied the comparative intubating conditions after remifentanil-propofol and alfentanil-propofol without neuromuscular blocking agents. He concluded that the combination of remifentanil 4 µg/ kg and propofol 2.5 mg/kg provided satisfactory intubating conditions without eliciting any cardiovascular response.

Jabbour-Khoury[12] et al (2003) in their study found that alfentanil produced better intubating conditions than fentanyl both of which were used in combination with lidocaine and propofol in the absence of muscle relaxants. He concluded that Propofol-Fentanyl combination could be used as an alternative technique for tracheal intubation in patients contraindicated to neuromuscular blocking agents.

Trabold et al[29] (2004) studied a combination of Sevoflurane, N_2O and remifentanil and concluded that this combination provided acceptable conditions for tracheal intubation in children and could be used as an acceptable alternative to intravenous induction and neuromuscular blockers.

Woods et al[31] (2005), studied the tracheal intubation with propofol and fentanyl, remifentanil, alfentanil without the usage of any neuromuscular blocking agents.

Prakash et al[20] (2006) showed that in the absence of neuromuscular blocking agents, better intubation results were produced by the combination of fentanyl-midazolam-propofol, when compared to fentanyl-lidocaine-propofol.

Mohammadreza safavi, Azim honarmand et al[18] (2008) conducted a randomized study of tracheal intubation by remifentanil or alfentanil in combination with thiopentone sodium in absence of muscle relaxants. The study showed remifentanil 4µg/kg or alfentanil 40µg/kg with thiopentone sodium 5mg/kg provided good to excellent intubating conditions without using neuromuscular blocking agents.

AIM

To compare the intubating conditions and cardiovascular changes (post induction) between fentanyl, midazolam, propofol and fentanyl, lignocaine, propofol groups without using neuromuscular blocking agents.

MATERIALS AND METHODS

It is a prospective double blind randomized controlled study. The study was approved by the ethical Committee.

Hundred patients undergoing elective general surgical procedure under general anaesthesia with endotracheal intubation were included in this study and randomly divided into two groups.

The Surgeons were duly informed about the study. The study was during the period of April 2011 to April 2012 in the Department of Anaesthesiology, Chengalpattu MedicalCollege, and Chengalpattu.

Group (M)

Fifty patients received propofol 2.5mg/kg, fentanyl 2µg/kg, midazolam 0.03mg/kg.

Group (L)

Fifty patients received propofol 2.5mg/kg, fentanyl 2µg/kg, lidocaine 1.5mg/kg.

Inclusion Criteria

ASA I&II

Age 20-50yrs

All cases requiring GA

Exclusion Criteria

Not meeting inclusion criteria

Known and difficult airways

Patients with full stomach

Patients posted for emergency surgery

Hypertension,

Diabetes,

Ischemic heart disease

Reactive Airway Disease

Allergy to drugs

Randomization was done by draw of lots. The follow up of the Patient and analysis of data were done by personnel blinded to which group belonged to. Drawing of lots for Randomization and preparation of study was prepared by a consultant who took no further part in the study, the anaesthetist performing and scoring the laryngoscopy grading and tracheal intubation was blinded to the randomization group and the rest of the study was conducted by investigator who was blinded to the drug injected.

MATERIALS

1. Inj.Profopol 1% - 10 ml vial
2. Inj.Fentanyl Citrate – 2ml ampoule
3. Inj.Glycopyrrolate – 1ml ampoule
4. Inj.Midazolam – 5ml vial
5. Inj.Lignocaine Hydrocholoride (xylocard) - 50 ml vail
6. Disposable 5ml syringes
7. McIntosh Laryngoscope with 3 and 4 size blades
8. Endotracheal tubes of varying sizes
9. Emergency drugs
10. Difficult Intubation Strategies

PRE OPREATIVE PREPARATION

All the Patients were admitted and they underwent relevant investigations. Preoperatively informed, written consent was obtained from the Patients.

Complete Hemogram,

Bleeding time,

Clotting time

Blood - urea, sugar

Serum - creatinine

Serum - Electrolytes

X ray Chest

Electrocardiogram

Other relevant investigations were obtained on the basis of the conditions of the Patient

ANAESTHESIA PROTOCOL

Pre operative visit was done to allay anxiety and good rapport was established with the patient.

All the patients were given pre operative night sedation with tablet diazepam 10mg and tablet ranitidine 150mg orally.

PREMEDICATION

All the patients were premedicated with Inj.Glycopyrrolate 4µg/kg body weight intramuscularly45 mins before surgery. Basal pulse rate and Blood Pressure were recorded.

MONITORING

Non Invasive Automated BP

Electrocardiogram

Pulseoximetry

ETCO2

Neuromascular Monitoring

Patients shifted to operating table after 45 minutes. In the operating room patients were connected to baseline monitors, then intravenous access established with 18 gauges cannula and intravenous fluids started. Pulse rate, Blood pressure, ECG and SpO2 were recorded.

PRE-OXYGENATION

Pre-oxygenation was done with 100% oxygen for 5 minutes.

ADMINISRATION OF STUDY DRUG

Patients In M Group received propofol 2.5mg/kg, fentanyl 2 µg/kg, midazolam 0.03 mg/kg, and; L group received propofol 2.5mg/kg, fentanyl 2 µg/kg, lignocaine 1.5 mg/kg. Fentanyl and midazolam were administered 5 min and lignocaine 20 s before induction of anaesthesia with propofol.

After loss of response to command the patient's lungs were ventilated via a anatomical facemask. Laryngoscopy was done 40 s after propofol administration. The patient's trachea was intubated with an appropriate size cuffed tracheal tube and the cuff was inflated. Anaesthesia was maintained with 66% nitrous oxide in oxygen and 0.6% isoflurane using a carbondioxide absorption circuit. After intubation the haemodynamic measurements were obtained up to 5mins of post intubation period.

The whole intra operative and post operative period were uneventful.

Summarized protocol

TABLE.VII

Time	M Group	L Group
-5 min	Fentanyl + Midazolam	Fentanyl + lignocaine 20 sec before induction
0	Propofol	Propofol
+ 40 s	Laryngoscopy and tracheal intubation	Laryngoscopy and tracheal intubation

ASSESSMENT OF INTUBATION CONDITIONS

Assessment of intubating conditions include: ease of laryngoscopy, the vocal cord position, the cough, and limb movement. Laryngoscopy was graded as easy (jaw relaxation), fair (jaw not fully relaxed), and difficult (poor jaw relaxation). Intubating conditions were regarded as excellent (all qualities were excellent), good (all qualities were either excellent or good), and poor (the presence of a single quality listed under poor). Excellent and good intubating conditions are comes under clinically acceptable; poor intubating conditions were regarded as clinically not acceptable

TABLE.VIII

Variable	Intubation conditions		
	Clinically acceptable		Not acceptable
	Excellent	Good	Poor
Laryngoscopy	Easy	Fair	Difficult
Vocal cords position	Abducted	Intermediate	Closed
Coughing	None	Diaphragm	Sustained (> 10 s)
Movement of the limbs	None	Slight	Vigorous

STATISTICAL ANALYSIS

Heartrate, mean arterial pressure, intubating conditions score include laryngoscopy, limb movement, vocal cord position, coughing are compared. All recorded data were entered SPSS 16.0V Software for determining the statistical significance. Mean and standard deviation for continuous variable and Percentages are given for categorical variables. Student's t test was used to compare the two groups on mean values of various parameters. Chisq test was used to compare the two groups for categorical variables. P value taken for significance is <0.05.

LIST OF SURGICAL PROCEDURES

TABLE.IX

S.No	Surgery	M Group	L Group
1	Herniorraphy	15	15
2	Fibroadenomaexicision	12	10
3	Appendicectomy	4	8
4	Gynaecomastia Exicision	3	2
5	Hydrocele eversion	6	5
6	Cholecystectomy	2	2
7	Epigastric hernia	4	4
8	Incisional hernia	4	4

OBSERVATION AND RESULTS

Hundred patients under this study were categorized into two groups. They comprised of both sexes with age ranging from 20-50 years

The age and sex were equal in all two groups. P value was not significant in the study done (P value is more than 0.05).

DEMOGRAPHIC PROFILE

AGE

The range of age in both group M and group L was 20 – 50 years. The average age in both groups was similar. The table describes the distribution of age.

TABLE - X

GROUP	20-30	31-40	41-50	TOTAL
	N (%)	N (%)	N (%)	N (%)
M Group	30 (60%)	13 (26%)	7 (14%)	50 (100%)
L Group	20 (40%)	18 (36%)	12 (24%)	50 (100%)
TOTAL	50	31	19	100

MEAN AGE OF PATIENTS BY GROUPS

The mean age group for M was 31.44±8.68 and L group 34.52±8.82 there is no statistically significant difference between M and L age group, with the P value 0.08.

SEX

This table shows the sex distribution of M and L groups. There is no significant difference between M and L group, p value - 0.52.

TABLE - XI

Group	Male N (%)	Female N (%)	Total	P value
M	32	18	50	0.52
L	35	15	50	
TOTAL	67	33	100	

WEIGHT

This table shows the mean weight of the patients in these two groups. Mean value of M group is 49.82±6.157, Mean value of L group is 52.15±6.63. The P value is 0.08

TABLE - XII

Group	N	Mean ± SD	Minimum weight (kgs)	Maximum weight (kgs)	P value
M	50	49.82 ± 6.157	32	64	0.08
L	50	52.15 ± 6.63	36	62	

HEIGHT

This table shows the mean height of the patients in these two groups. The height for M group 144cms to 170cms. The mean height for M group is 159.32±6.912.The height for L group 148cms to 168cms. The mean height for L group is 160.68±6.31.There is no significant difference between M and L group for height with t value of 1.028 and also P value of 0.31.

TABLE – XIII

Group	N	Mean ± Std. Deviation	Minimum Height (cms)	Maximum Height (cms)	P value
M	50	159.32±6.912	144	170	0.31
L	50	160.68±6.31	148	168	

Mallampatti Grading (MPC I/II)

Airway was assessed by using the Mallampatti grading. Airways of both group were compared and In M group 44 (88%) patients, In L group 45(90%) patients comes under MPC grade –I, In M group 6 (12%) patients, In L group 5(10%) patients comes under MPC grade –II. chisq value for this grading 0.012, P value is 0.75.There is no statistical significance between these two groups.

TABLE - XIV

Group	Grade	Frequency (N)	Percentage (%)	Total	P value
M	I	44	88	50	0.75
	II	6	12	100	
L	I	45	90	50	
	II	5	10	100	

Cormack and Lehane Laryngoscopic view (CLG I/II):

This classification describes the best view during laryngoscopy, Laryngoscopy view of both groups were compared and In M group 45 (90%) patients, In L group 47(94%) patients comes under CLG grade–I, In M group 5 (10%) patients, In L group 3(6%) patients comes under MPC grade–II. Chisq value for this grading 0.54, P value is 0.4. There is no statistical difference between two groups.

TABLE - XV

Group	Grade	Frequency (N)	Percentage (%)	Total	P value
M	I	45	90	50	0.4
	II	5	10	100	
L	I	47	94	50	
	II	3	6	100	

Duration Laryngoscopy (S)

Duration of the laryngoscopy is defined as the time from start of laryngoscopy until tracheal intubation and removal of laryngoscope blade from the mouth. Laryngoscopy was performed 40 sec after propofol administration, maximum laryngoscopy duration in M group 17sec, L group is 19sec, minimum duration of both groups are respectively 11secs (M), 12 secs (L). Mean laryngoscopy duration of both groups are respectively 13.62secs (M), 15.4secs (L). Duration of laryngoscopy was statistically significant between these two groups.

TABLE – XVI

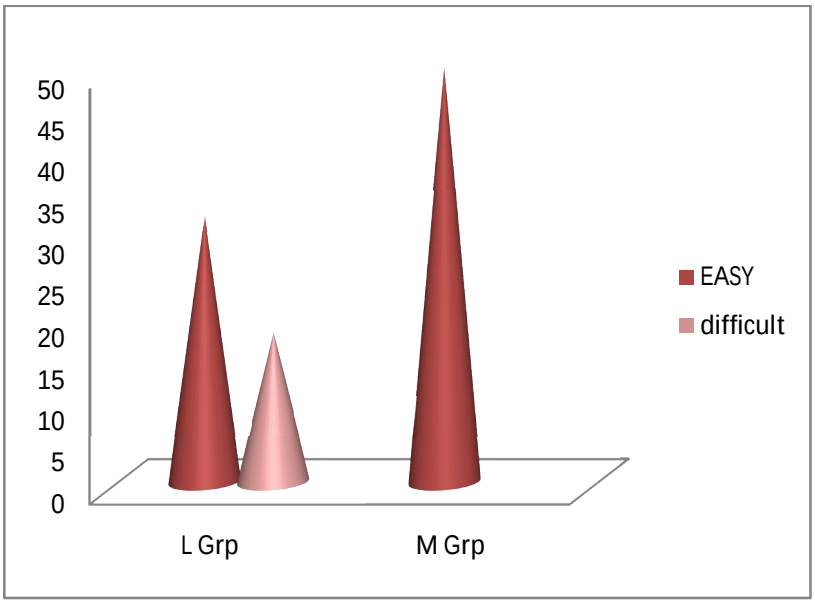
Group	N	Mean±Std. Deviation (S)	Minimum Duration (S)	Maximum Duration (S)	P value
M	50	13.62±1.652	11	17	0.00
L	50	15.4±2.1	12	19	

LARYNGOSCOPY

Laryngoscopy is graded as easy, fair and difficult. Easy, fair comes under clinically acceptable intubating conditions. Difficult laryngoscopy comes under clinically unacceptable intubating condition. In M group laryngoscopy was easy 50 (100%) in all patients whereas in L group 32(64%) had easy laryngoscopy, 18(36%) had difficult laryngoscopy. P value is 0.00(less than 0.05). M group had better laryngoscopy than L group.

TABLE - XVII

Group	Grade	Frequency (N)	Percentage (%)	P value
M	Easy	50	100	0.00
L	Easy	32	64	
	Difficult	18	36	

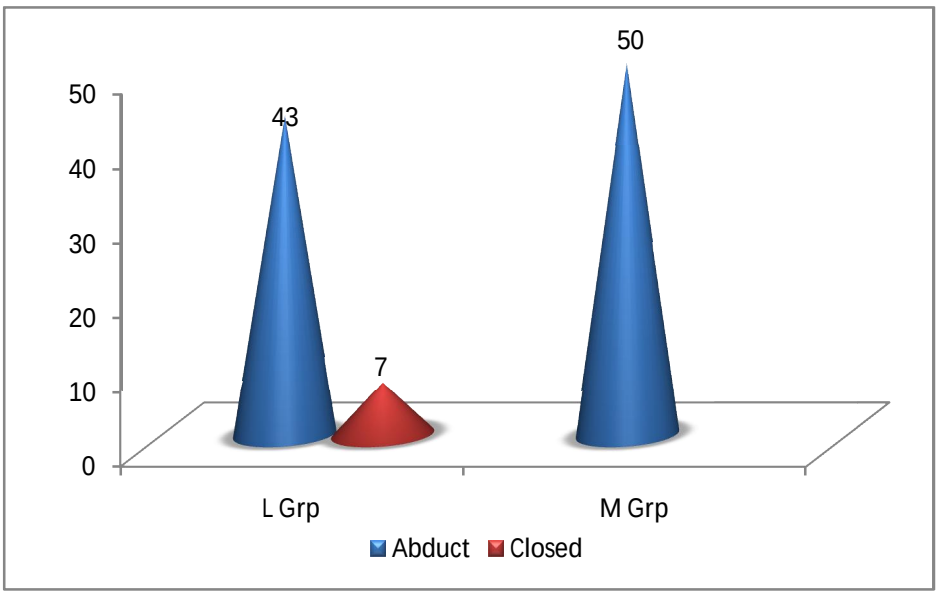


Vocal Cord

During laryngoscopy vocal cord position is classified as abducted, intermediate (clinically acceptable intubating condition), closed(clinically unacceptable intubating condition).Vocal cord position is abducted 50 out of 50 patients in M group,43 out of 50 patients in L group .Seven patients in the L group had closed vocal cords requiring administration of rocuronium before successful intubation.

TABLE - XVIII

Group	Vocal cord position	Frequency (N)	Percentage (%)	Rocuronium Requirement	p Value
M	Abduct	50	100	0	0.006
L	Abduct	43	86	0	
	Closed	7	14	7	

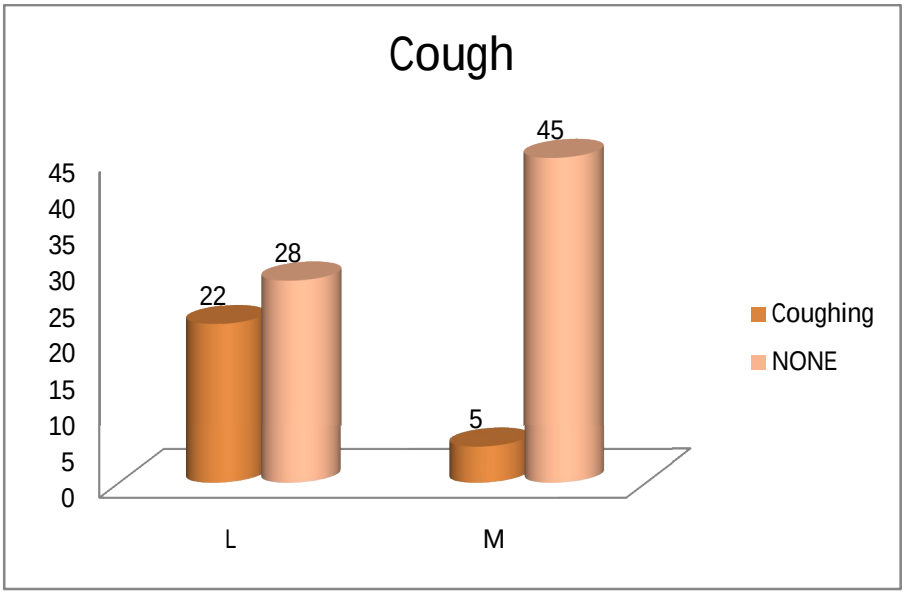


Cough

During laryngoscopy and intubation, coughing is graded as no cough, diaphragmatic cough (clinically acceptable), sustained coughing more than 10secs (clinically unacceptable). Twenty two patients (44%) in the L group had sustained coughing (> 10 s) on intubation compared with the M group 5(10%) although this is statistically significant. P value is 0.00

TABLE - XIX

Group	Cough	Frequency (n)	Percent (%)	P value
M	Coughing	5	10	0.00
	None	45	90	
	Total	50	50	
L	Coughing	22	44	
	None	28	28	
	Total	50	50	

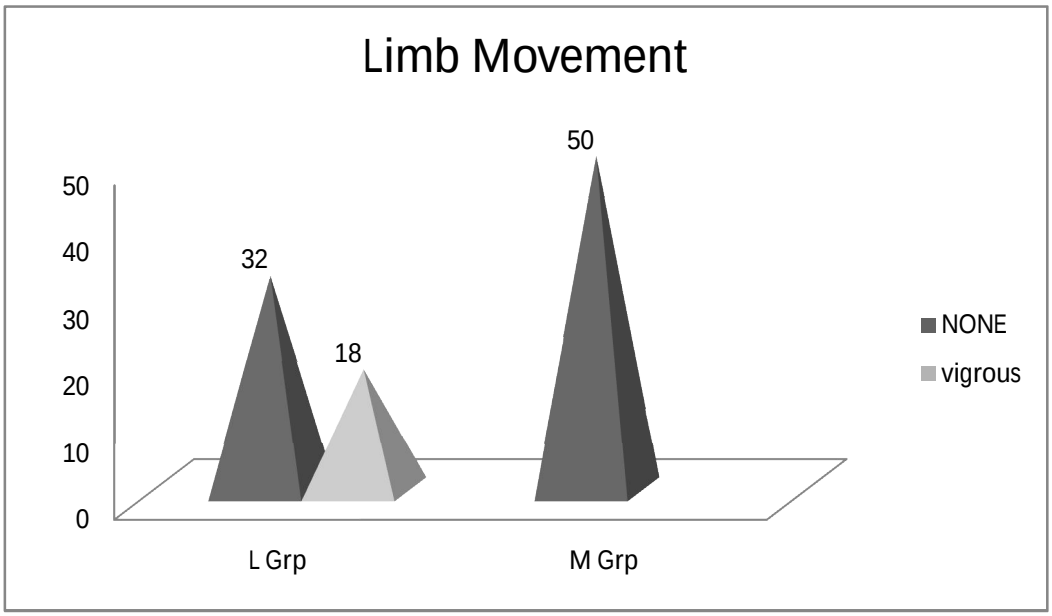


LIMB MOVEMENTS

Limb movement is graded as no movement, slight movement **(**clinically acceptable), vigorous movement (clinically unacceptable). In the L group 18 patients (36%) had vigorous limb movement compared with no limb movement in the M group (P value is 0.00).

TABLE - XX

Group	Limb movement	Frequency (n)	Percentage (%)	P value
M	Limb movement (none)	50	100	0.00
L	Limb movement (none)	32	64	
	Vigorous	18	36	



Overall intubating condition

This table shows the overall intubating condition of both groups. M group shows better intubating conditions 40 out of 50 patients (80%), compare to 28 out of 50 patients (56%) in L Group. Clinically UN acceptable intubating conditions in both groups respectively 10(20%) in M (g), 22(44%) in L (g). There is no rocuronium requirement in M group. In L group seven are required rocuronium, over all intubating conditions better in M group.

TABLE - XXI

Group	Intubating conditions	Frequency (n)	Percentage (%)	Total	Rocuronium Requirement	P value
M	CA	40	80	50	0	0.01
	C-UA	10	20	100		
L	CA	28	56	50	7	
	C-UA	22	44	100		

CA – Clinically acceptable C-UA – Clinically unacceptable

HEART RATE (BEAT / MINUTE)

The maximum rise in heart rate was observed during intubation and at one minute following intubation. The rise was modest of about 2-3 beats per minute. It started declining to baseline values at fourth minute. There was a further decline in heart rate from the baseline values at sixth and seventh minute. No rhythm disturbances were observed. There is no significant difference in heart rate of both groups. P value is not clinically significant.

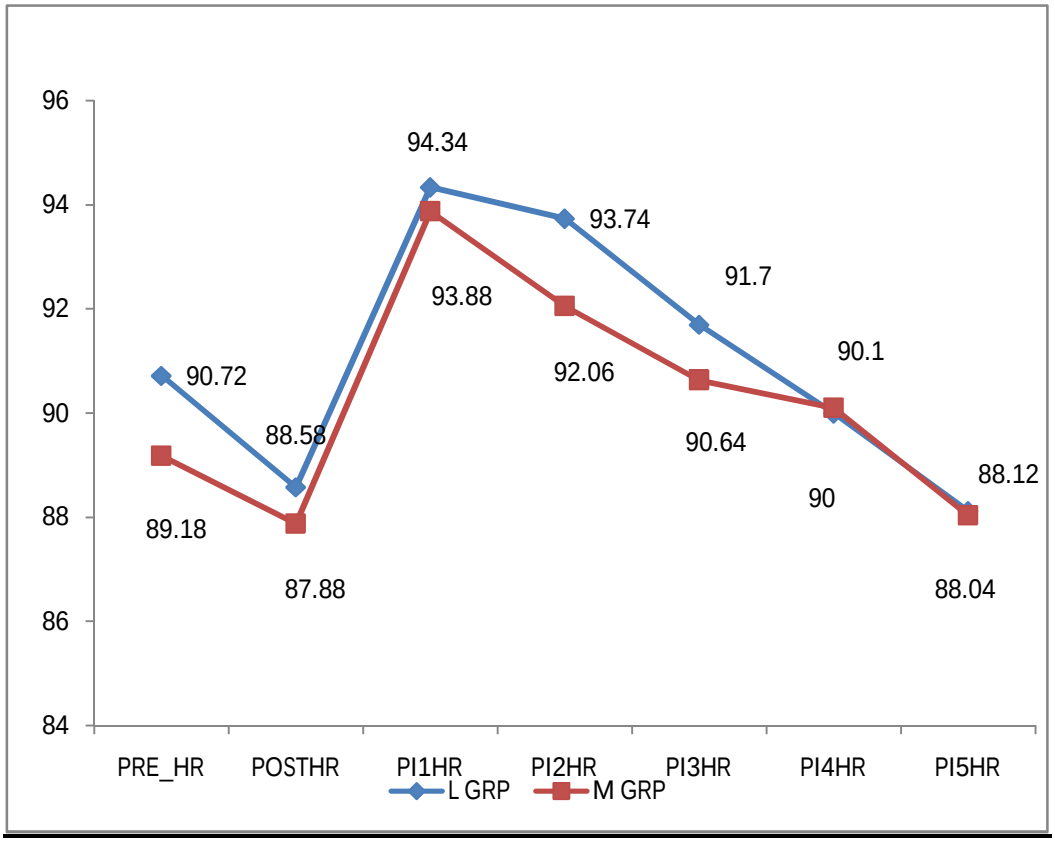


TABLE – XXII

DISTRIBUTION OF MEAN ± STANDARD DEVIATION OF HEART RATE BY GROUPS

Events	M Group (n = 50)	L Group (n = 50)	P Value
Pre-induction	89.18 ± 3.56	90.72 ± 5.59	0.10
Post-induction	87.88 ± 3.9	88.58 ± 4.03	0.38
POST – INTUBATION			
1 min	93.88 ± 6.48	94.34 ± 4.44	0.68
2 min	92.06 ± 6.27	93.74 ± 4.36	0.12
3 min	90.64 ± 6.56	91.7 ± 4.23	0.34
4 min	90.1 ± 6.49	90 ± 4.31	0.93
5 min	89.26 ± 6.24	88.12 ± 4.36	0.94

MEAN ARTERIAL PRESSURE (mm/Hg)

Statistical significance is not observed between the two groups up to induction. (P value more than 0.05). After induction there is significant difference on mean arterial pressure between these two groups till the end of the study. (P value less than 0.00). Between these two groups better hemodynamic stability was observed in M group.

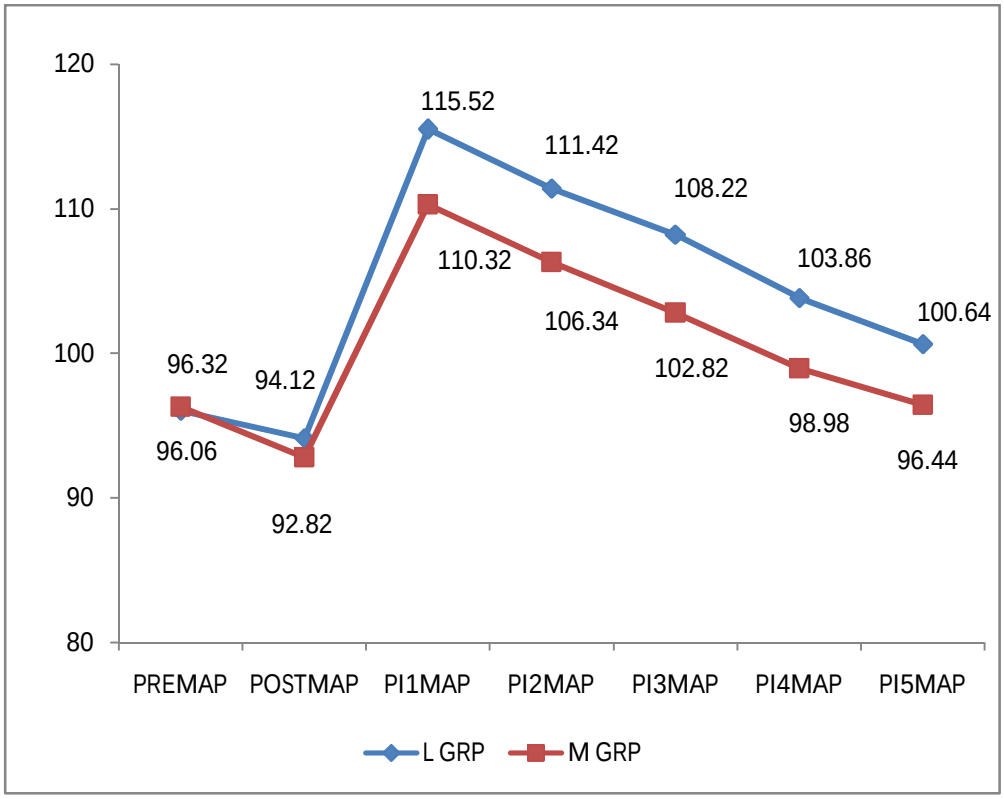


TABLE -XXIII

DISTRIBUTION OF MEAN ± STANDARD DEVIATION OF MEAN ARTERIAL PRESSURE BY GROUPS

Events	M Group (n = 50)	L Group (n = 50)	P Value
Pre –Induction	96.32 ± 4.98	96.06± 4.86	0.79
Post –Induction	91.7 ± 5.43	94.12 ±491	0.02
POST INTUBATION			
1 min	110.32 ± 3.9	115.52 ±4.47	0.00
2 min	106.34 ± 4.52	111.42 ± 4.53	0.00
3 min	102.82 ± 4.05	108.22 ±4.59	0.00
4 min	98.98 ± 4.09	103.86± 4.61	0.00
5 min	96.44 ± 3.53	100.64 ±3.93	0.00

RESULTS

There is no statistical significance in patient characteristics between these two groups (Table X to XIII). The Mallampatti grading, Cormack and lehane laryngoscopy grading, and the laryngoscopic duration was compared in both groups. There is no statistical significance in mallampatti and Cormack – lehane grading between these two groups, the statistical significance was observed in laryngoscopy duration (P value is 0.00).

Mask ventilation was easy in all patients. Intubation attempt was successful in all (100%) patients in the M group and in the L group 43 out of 50 patients (86%) had successful intubation. There is no rocuronium requirement in the M group and in the L group seven patients required rocuronium (P value 0.01). Patients who are all received rocuronium were intubated successfully.

Overall clinically acceptable intubating conditions was 40 out of 50 patients (80%) in the compared to 28 out of 40 patients (56%) in L group. This difference was statistically significant (P value 0.01) (Table 11). Laryngoscopy was easy in all patients in the M group. Laryngoscopy was difficult in 18 (36%) out of 50 patients in the L group (P value 0.00). seven patients in the L group had closed vocal cords requiring administration of rocuronium before intubation (P value 0.01).

Twenty two patients (44%) in the L group had sustained coughing (> 10 s) on intubation compared with the M group 5(10%) although this is statistically significant. P value is 0.00

In the L group 18 patients (36%) had vigorous limb movement compared with no limb movement in the M group (P value is 0.00).

Cardiovascular responses to induction and intubation are compared in both groups. There is no significant difference in heart rate of both groups. Statistical significance is not observed between the two groups up to induction (P value more than 0.05). After induction there is significant difference on mean arterial pressure between these two groups till the end of the study (P value less than 0.00). Between these two groups better hemodynamic stability was observed in M group.

Oxygen saturation was maintained between 96% - 100% before as well as after induction of anaesthesia and tracheal intubation. There were no episodes of laryngospasm, bronchospasm, masseter spasm, or generalized rigidity were observed.

DISCUSSION

Endotracheal intubation without usage of neuromuscular blocking agents is successfully described in this study. This technique enables us to assess the airway by laryngoscopy to ascertain if oxygenation is possible. This technique is useful in both expected and unanticipated difficult intubation, in cases where muscle relaxants are either contraindicated (e.g. myopathies) or not required to facilitate surgical access.

Demographic profile

The two groups were comparable with respect to age, sex, height and weight (Table X to XIII) in our study and are correlating with study done by prakash et al[20] whose study also shows no statistical difference in patient characters.

Mallampatti grading

Airways of both group were compared and 44 patients (88%) in the M group, 45 patients (90%) in the L group comes under MPC grade – I, 6 patients (12%) in the M group , 5 patients(10%) in the L group comes under MPC grade –II. There is no statistical significance between these two groups. Prakash et al[20] his study also showed no statistical difference between two groups in Mallampatti grading.

Cormack and lehane laryngoscopy grading

Laryngoscopy view of both groups were compared and 45 patients (90%) in the M group, 47 patients (94%) in the L group comes under CLG grade–I, 5 patients (10%) in the M group, 3 patients (6%) in the L group comes under MPC grade–II. Thus the two groups did not differ statistically with respect to laryngoscopic view.

Prakash et al[20] study which also no statistical difference between two groups in mallampatti grading.

Laryngoscopic duration

Duration of the laryngoscopy is defined as the time from start of laryngoscopy until tracheal intubation and removal of laryngoscope blade from the mouth. Laryngoscopy was performed 40 sec after propofol administration, maximum laryngoscopy duration in M group duration was 17sec, and L group it is 19 sec, minimum laryngoscopy duration of both groups are respectively 11secs(M),12secs(L). Mean laryngoscopy duration of both groups are respectively 13.62secs (M), 15.4secs (L). Laryngoscopy duration was statistically significant between these two groups.

Prakash et al[20] showed no statistical difference between two groups in laryngoscopy duration.

Laryngoscopy

In M group laryngoscopy was easy in all patients (100%) whereas in L group 32(64%) had easy laryngoscopy, 18(36%) had difficult laryngoscopy. It is statistically significant. In M group laryngoscopy was better than L group.

Prakash et al[20] study showed Laryngoscopy was easy in all patients in the M group but difficult in two out of 40 patients in the L group

Lewis et al[16] (1948) also showed in his study that there was difficulty in performing laryngoscopy in six patients. He used thiopental sodium as the sole agent to facilitate tracheal intubation without using neuromuscular blocking agents

Vocal cord position

It is abducted in 50 out of 50 patients in M group, 43 out of 50 patients in L group. In the L (lidocaine) group seven patients had closed vocal cords requiring rocuronium for intubation. It is statistically significant (P value 0.01). This is in concurrence with prakash et al[20] study which showed five patients in the L (lidocaine) group had closed vocal cords and so these patients required rocuronium for intubation.

Coughing

In our study twenty two patients (44%) in the L (lidocaine1.5 mg/kg) group had sustained coughing (> 10 s) on intubation compared with five patients (10%) in the M (midazolam) group. This is statistically significant. P value is 0.00.

Prakash et al[20] study showed sustained coughing (> 10 s) in the L group (17 patients) during intubation compared with the M group (10 patients).

Davidson et al[5] (1993) studied tracheal intubation with propofol, alfentanil and with or without intravenous lignocaine. His study showed better cough suppressant effect in lignocaine.

Houlton et al[19] (1979) study showed equal cough suppressant effect in lidocaine compared to bronchodilators.

Yukiola et al[32] (1985) study showed decreased incidence of cough when 2 mg/kg of intravenous lidocaine was given one to five minutes before intubation.

Hiller et al[10] (1993) study showed lignocaine 1 mg/kg not enough to suppress the cough. He concluded that higher dose of lignocaine was required to suppress the cough reflex with propofol induction**.**

Limb movement

In the L group 18 patients (36%) had vigorous limb movement compared with no limb movement in the M group (P value is 0.00). This is in concurrence with Prakash et al[20] study which showed that six patients in the L (lidocaine) group had slight limb movement compared with no limb movement observed in the M group.

Intubating conditions

In our study M group shows better intubating conditions in 40 out of 50 patients (80%), compared to 28 out of 50 patients (56%) in L Group. Clinically unacceptable intubating conditions in both groups was respectively 10(20%) in M (g), 22(44%) in L (g). This difference was statistically significant (P value 0.01) (Table XXI). There is no rocuronium requirement in M group. In L group seven patients required rocuronium, and over all intubating conditions were better in M group.

Prakash et al[20] study showed better intubating conditions in midazolam group compared to lignocaine group.

Lewis et al[16] (1948) studied after administration of thiopentone sodium 500-750 mg in 200 patients for oral intubation or blind nasal intubation without muscle relaxants. There were two failures in the blind nasal group.

Keaveny et al[13] (1988) in his study used propofol 3 mg/kg and showed better intubating conditions.

Barker et al[2] (1992) study showed lower incidence of laryngospasm and vocal cord movements following propofol induction compared to thiopentone induction, and is due to greater depression of laryngeal reflexes by propofol.

Grant et al[7] (1998) study showed better intubating conditions with propofol 2 mg/kg and pre treatment with remifentanil 2 µg/kg. This dose was equal to 4 µg/kg of fentanyl.

Mulholland et al [17] (1991) study showed no significant difference was found in the intubating conditions with intravenous pre-treatment with lignocaine 1.5 mg/kg.

Grange et al[6] (1993) observed no significant difference in the quality of intubating conditions with intravenous pre-treatment with lignocaine or alfentanil.

Klemola et al[15] (2000) study showed better intubating conditions was observed in remifentanil 4µg/kg –propofol 2.5 mg/kg comination.

Trabold et al[29] (2004) study showed better intubating conditions was observed when remifentanil 1µg/kg was given after propofol 2.5 mg/kg with midazolam 0.03 mg/kg.

HEART RATE

In our study cardiovascular responses to induction and endo tracheal intubation were compared with midazolam and lidocaine groups. In both groups no difference in heart rate was found. (P value more than 0.05).This is in concurrence with prakash et al[20] study that showed that there is no statistical significance in heart rate between two groups.

Mulholland et al [17] (1991) study showed no difference in the heart rate to intubation with propofol (2.5 mg/kg) induction with pre-treatment with lignocaine 1.5 mg/kg.

Mean arterial pressure

In our study there was significant difference in mean arterial pressure after induction between these two groups till the end of the study. (P value less than 0.00). Between these two groups better cardiovascular stability was observed in M group. This is in concurrence with Prakash et al[20] study that showed that there was statistical significance in mean arterial pressue between two groups. In his study better cardiovascular stability was observed in M group compared to L group.

Saarnivaara et al[26] (1991) study showed better cardiovascular stability in propofol 2.5 mg/kg with alfentanil 30 µg/kg pretreatment.

Mulholland et al [17] (1991) study showed no difference in the mean arterial pressure to intubation with propofol (2.5 mg/kg) induction with pre-treatment with lignocaine 1.5 mg/kg.

Klemola et al[15] (2000) study showed better cardiovascular stability was observed in remifentanil 4µg/kg with propofol 2.5 mg/kg combination.

Trabold et al[29] (2004) study showed better cardiovascular stability was observed in remifentanil 1µg/kg was given after s 2.5 mg/kg with midazolam 0.03 mg/kg.

SIDE EFFECTS

In our study there were no episodes of laryngospasm, bronchospasm, masseter spasm, or generalized rigidity. This is in concurrence with Lewis et al[16] study which showed that problems like coughing, laryngospasm occur during thiopentone induction alone without using neuromuscular blocking agents.

The propofol (2.5 mg/kg) induction has greater depression of laryngeal reflexes than an equipotent dose of thiopentone. The incidence of laryngospasm was lower with propofol compared to thiopentone.[13]

The addition of fentanyl and midazolam potentiate the effects of propofol and reduce the dose requirement of propofol. Both propofol and midazolam has synergestic action due to interaction at GABA-A receptors in the central nervous system. The propofol dose was reduced by 52% in the presence of midazolam.[27]

Midazolam has synergistic action with fentanyl for induction of anaesthesia. This synergistic effect is due to potentiation between opioids and benzodiazepines.[1]

The cough suppressant effect of intravenous lignocaine is due to brain stem depression.[11] Lignocaine may act by anaesthetizing peripheral cough receptors in the trachea and hypopharynx[19] or by increasing the depth of general anaesthesia[8].

Endotracheal intubation is a stronger stimulus than laryngoscopy. Propofol with fentanyl combination was able to suppress motor and hemodynamic reactions to various noxious stimuli. Laryngoscopy was easier in most of the patients with either technique.[14]

The tracheal intubation without neuromuscular blocking agents are not advised in patients with a full stomach, elderly patients and those with cardiovascular or cerebrovascular disease and in those patients undergoing neurosurgery or ophthalmic procedures.

The potentially serious and undesirable side-effects of succinylcholine are avoided and side effects such as anaphylaxis that can occur with the use of non-depolarizing drugs are avoided. The short acting opioids, such as remifentanil and alfentanil, when used in combination with propofol for tracheal intubation are more advantageous in the aspect of good depth of anaesthesia and also stable hemodynamic profile .[31] In our study we used fentanyl as a opioid in combination with

midazolam, propofol and lidocaine. Midazolam has synergestic action with fentanyl and propofol. So, the intubating condions and cardiovascular responses were better in propofol, midazolam and fentanyl group patients.

CONCLUSION

I conclude that the propofol – fentanyl – midazolam combination is better compared to propofol – fentanyl – lignocaine combination in providing clinically acceptable conditions for intubation without significant cardiovascular changes without the use of neuromuscular blocking agents. Hence this combination can be a useful alternative technique for tracheal intubation when neuromuscular blocking drugs are contraindicated or need to be avoided.

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PROFORMA

NAME: AGE/ SEX: IP.NO:

WT: HT:

DIAGNOSIS: SURGERY:

PRE OP ASSESSMENT

INVESTIGATIONS

Hb%	BT	CT	Ecg	CxR	Blood.urea	Sr.creatinine

MONITORS

ECG	NIBP	SPO2

AIRWAY ASSESSMENT :

MALLAMPATTI CLASSIFICATION :

CORMACK & LEHANE CLASSIFICATION :

PRE MEDICATION :

INDUCTION :

INTUBATION :

MAINTAINANCE :

DURATION OF LARYNGOSCOPY :

INDUBATING CONDITIONS

Time Point	Mean Arterial Pressure	Heart Rate	Intubating Conditions		
			Not Acceptable	Clinically Acceptable	Rocuronium Requirement
Pre induction					
Post induction					
Post Intubtion					
1 min					
2 mins					
3 mins					
4 mins					
5 mins					

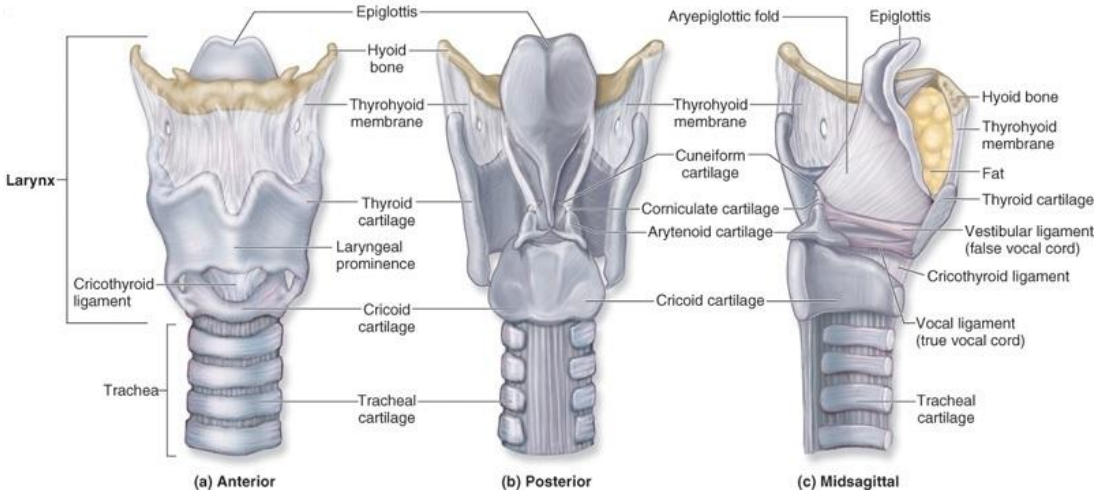
M GROUP

S. No	IPNO	Age	WT (KG)	HT (CM)	Sex	MPC VII	CLG VII	DURATION LARYNGOSCOPY	INDUBATING CONDITION						PRE INDUCTION		POST INDUCTIONP		POST INDUBATION									
									LARYN GOSCOPY	VOCAL CORDS position		LIMB MOVMENTS	CA\-C-UA	Rocuronium Requirement					1 MIN		2 MIN		3 MIN		4 MIN		5 min	
															HR	MAP	HR	MAP	HR	MAP	HR	MAP	HR	MAP	HR	MAP	HR	MAP
1.	39712	49	54	162	M	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	91	99	88	96	99	113	97	108	95	105	95	100	94	98
2.	37756	40	55	151	F	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	92	104	87	100	96	109	96	105	93	101	91	98	90	95
3.	39935	50	60	165	M	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	89	102	86	99	97	114	95	110	94	106	93	104	92	101
4.	39585	40	55	163	M	II	I	15	EASY	ABDUCT	NONE	NONE	CA	No	92	98	89	95	99	111	97	107	96	104	95	100	94	98
5.	39882	48	52	167	M	I	I	13	EASY	ABDUCT	Coughing	NONE	C-UA	No	102	85	98	82	108	102	103	96	105	93	104	90	103	88
6.	39736	47	55	157	M	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	88	94	84	90	94	112	92	108	91	104	90	101	88	98
7.	39788	27	38	160	M	I	II	14	EASY	ABDUCT	NONE	NONE	CA	No	90	98	94	94	91	110	89	108	87	104	87	101	86	98
8.	36928	25	50	156	M	I	I	16	EASY	ABDUCT	NONE	NONE	CA	No	91	101	90	96	90	108	88	106	86	103	85	99	85	96
9.	49420	26	50	149	F	I	I	17	EASY	ABDUCT	NONE	NONE	CA	No	90	92	92	90	85	106	83	101	82	98	80	96	80	93
10.	48433	28	39	159	M	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	86	93	93	90	87	111	85	106	84	102	84	98	84	95
11.	39933	25	50	164	M	I	I	11	EASY	ABDUCT	NONE	NONE	CA	No	92	96	90	92	84	114	82	109	80	106	98	101	78	99
12.	40533	20	50	150	F	I	I	13	EASY	ABDUCT	NONE	NONE	CA	No	88	92	86	92	95	109	95	105	92	103	91	98	90	95
13.	40439	20	48	152	F	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	90	100	88	86	96	106	94	102	92	99	89	96	88	94
14.	40887	30	55	163	M	I	I	14	EASY	ABDUCT	Coughing	NONE	C-UA	No	90	93	88	90	96	110	94	106	95	102	94	98	93	96
15.	41244	27	38	169	M	I	I	15	EASY	ABDUCT	Coughing	NONE	C-UA	No	86	106	85	100	84	120	82	114	81	110	82	105	80	104
16.	41219	26	45	152	F	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	89	101	92	97	90	115	88	116	87	106	86	103	85	100
17.	41728	27	36	154	F	I	I	16	EASY	ABDUCT	NONE	NONE	CA	No	91	101	89	96	88	118	86	118	85	110	85	106	84	102
18.	41616	35	48	155	F	I	I	17	EASY	ABDUCT	NONE	NONE	CA	No	88	102	87	96	96	112	94	108	93	104	92	100	90	96
19.	42298	29	50	165	M	II	II	12	EASY	ABDUCT	NONE	NONE	CA	No	88	97	86	94	98	110	98	106	95	102	94	99	93	95
20.	41983	22	45	156	F	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	87	102	81	100	83	118	81	113	80	109	78	105	98	101
21.	42558	20	45	144	F	I	I	13	EASY	ABDUCT	NONE	NONE	CA	No	87	97	86	94	99	113	97	108	95	105	95	100	94	98
22.	42548	24	46	169	M	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	88	96	88	94	96	109	94	105	93	101	91	98	90	95
23.	50313	33	55	165	M	I	I	15	EASY	ABDUCT	NONE	NONE	C-UA	No	86	92	86	90	97	114	95	110	94	106	93	104	92	101
24.	42385	40	56	167	M	I	I	17	EASY	ABDUCT	NONE	NONE	CA	No	90	85	86	80	99	111	99	107	96	104	95	100	94	98
25.	42552	48	53	163	M	II	II	14	EASY	ABDUCT	Coughing	NONE	C-UA	No	91	98	90	92	108	102	105	96	105	93	104	90	103	88
26.	43033	25	49	152	F	I	I	13	EASY	ABDUCT	NONE	NONE	CA	No	86	92	84	90	94	112	92	108	91	104	90	101	88	98
27.	47209	27	52	147	F	I	I	16	EASY	ABDUCT	NONE	NONE	CA	No	88	93	86	82	91	110	90	108	87	104	87	101	86	98
28.	43222	31	56	164	M	I	I	12	EASY	ABDUCT	NONE	NONE	C-UA	No	82	100	80	84	90	108	88	106	86	108	86	99	85	96
29.	44075	35	52	152	F	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	86	98	86	94	85	106	83	101	82	98	80	96	80	93
30.	44082	22	47	156	F	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	90	96	89	90	87	111	87	106	84	102	84	98	84	95
31.	32186	30	51	150	F	I	II	12	EASY	ABDUCT	Coughing	NONE	C-UA	No	91	99	88	96	99	113	97	108	95	105	95	100	94	98
32.	32198	20	40	147	F	I	I	11	EASY	ABDUCT	NONE	NONE	CA	No	92	104	90	100	96	109	94	105	93	101	91	98	90	95
33.	32199	30	46	160	M	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	89	102	88	99	97	114	95	110	94	106	93	104	92	101
34.	32167	28	48	162	M	II	I	15	EASY	ABDUCT	NONE	NONE	CA	No	92	98	90	90	99	111	99	107	96	104	95	100	94	98
35.	32039	23	52	165	M	I	I	14	EASY	ABDUCT	NONE	NONE	C-UA	No	102	85	98	80	108	102	105	96	105	93	104	90	103	88
36.	30364	40	45	170	M	I	I	11	EASY	ABDUCT	NONE	NONE	CA	No	88	94	86	90	94	112	92	108	91	104	90	101	88	98
37.	31262	27	49	149	F	I	I	13	EASY	ABDUCT	NONE	NONE	CA	No	90	98	94	96	91	110	89	108	87	104	87	101	86	98
38.	30347	20	52	167	M	II	I	12	EASY	ABDUCT	NONE	NONE	C-UA	No	91	101	90	96	90	108	88	106	86	103	83	99	85	96
39.	29121	37	43	155	F	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	90	92	92	90	85	106	85	101	82	98	80	86	80	93
40.	30342	30	46	157	F	I	I	13	EASY	ABDUCT	NONE	NONE	CA	No	86	93	93	95	87	111	85	106	84	102	84	98	84	95
41.	38360	30	50	160	M	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	87	97	86	94	99	113	97	108	95	105	95	100	94	98
42.	38353	24	45	162	M	II	I	11	EASY	ABDUCT	NONE	NONE	CA	No	88	96	84	95	96	109	94	105	93	101	91	98	90	95
43.	38129	40	40	165	M	I	I	15	EASY	ABDUCT	NONE	NONE	C-UA	No	86	92	84	90	97	114	95	110	94	106	93	104	92	101
44.	30055	29	51	170	M	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	90	85	88	82	99	111	97	107	96	104	95	100	94	98
45.	37216	45	56	160	M	I	II	14	EASY	ABDUCT	NONE	NONE	CA	No	91	98	88	90	108	102	105	96	105	93	104	90	103	88
46.	36941	25	52	162	M	I	I	11	EASY	ABDUCT	NONE	NONE	CA	No	86	92	84	90	94	112	92	108	91	104	90	101	88	98
47.	35296	45	60	165	M	I	I	13	EASY	ABDUCT	NONE	NONE	CA	No	88	93	86	82	91	110	89	108	87	104	87	101	86	98
48.	35959	40	59	170	M	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	82	100	80	84	90	108	88	106	86	108	86	99	85	96
49.	35050	31	62	160	M	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	86	98	82	90	85	106	83	101	82	98	80	96	80	93
50.	34467	32	60	162	M	I	I	13	EASY	ABDUCT	NONE	NONE	CA	No	90	96	89	91	87	111	85	106	84	102	84	98	84	95

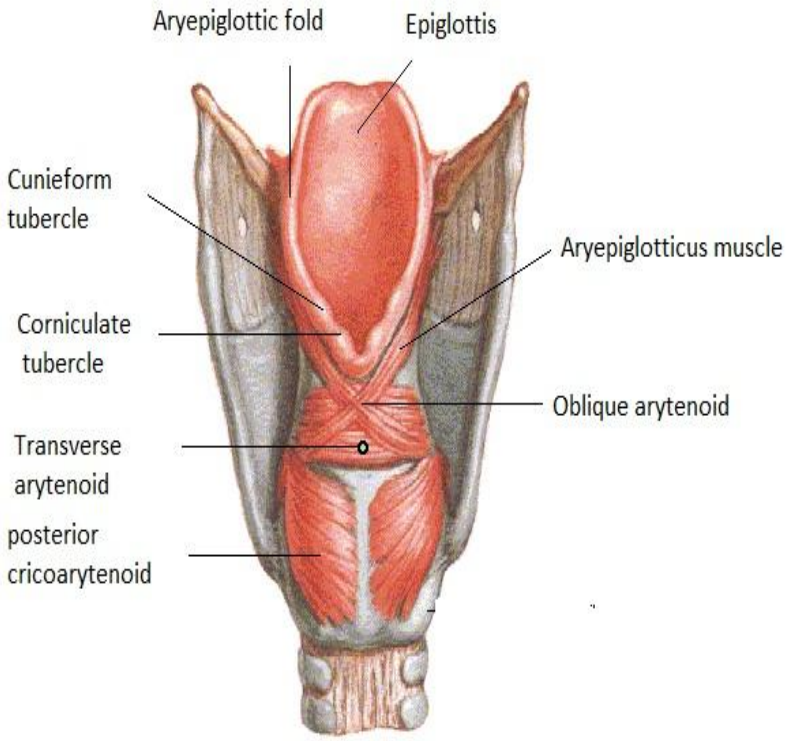
L GROUP

S.No	IPNO	Age	WT (KG)	HT (CM)	Sex	MPC VII	CLG VII	DURATION LARYNGOSCOPY	INDUBATING CONDITION						PRE INDUCTION		POST INDUCTION		POST INDUBATION									
									LARYN GOSCOPY	VOCAL CORDS position	COUGHING	LIMB MOVMENTS	CA\-C-UA	Rocuronium requirement					1 MIN		2 MIN		3 MIN		4 MIN		5 min	
															HR	MAP	HR	MAP	HR	MAP	HR	MAP	HR	MAP	HR	MAP	HR	MAP
1.	39041	35	56	161	M	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	85	98	87	98	100	120	90	112	98	109	97	104	95	103
2.	30416	28	51	151	F	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	90	97	90	97	93	120	90	113	91	109	91	103	88	100
3.	34380	45	58	162	M	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	85	98	84	90	90	115	90	112	88	108	86	105	84	100
4.	34950	28	59	164	M	I	I	17	difficult	closed	Coughing	vigrous	C-UA	No	88	103	88	99	94	119	92	115	91	111	89	110	87	106
5.	34947	26	60	166	M	I	I	18	EASY	ABDUCT	Coughing	NONE	C-UA	No	91	97	90	97	97	118	96	118	94	118	92	110	90	106
6.	35072	40	61	167	M	I	I	19	EASY	ABDUCT	NONE	NONE	CA	No	90	97	90	97	99	119	97	117	93	111	91	109	90	105
7.	35577	30	59	165	M	I	I	17	difficult	closed	Coughing	vigrous	C-UA	No	85	98	83	96	85	115	85	111	83	109	81	103	79	101
8.	35706	18	35	156	F	II	II	16	EASY	ABDUCT	NONE	NONE	CA	No	100	90	91	89	93	111	93	106	90	103	88	97	86	95
9.	35705	30	45	152	F	I	I	16	EASY	ABDUCT	NONE	NONE	CA	No	102	85	93	84	95	106	93	102	93	100	91	96	89	94
10.	33744	20	46	165	M	I	I	17	difficult	closed	Coughing	vigrous	C-UA	No	96	97	90	96	100	113	98	110	97	108	95	103	93	100
11.	36976	30	56	166	M	I	I	15	difficult	closed	Coughing	vigrous	C-UA	No	83	102	83	102	85	125	85	119	83	116	81	112	80	107
12.	35972	48	36	164	M	I	I	13	EASY	ABDUCT	NONE	NONE	CA	No	88	98	86	92	92	115	92	110	88	106	87	103	86	99
13.	36769	29	41	153	F	I	II	12	EASY	ABDUCT	NONE	NONE	CA	No	94	90	86	85	98	108	98	104	97	99	95	97	94	95
14.	40131	48	56	168	M	I	I	12	EASY	ABDUCT	Coughing	NONE	C-UA	No	90	98	90	97	93	115	93	110	94	108	92	103	90	100
15.	36854	30	51	152	F	I	I	14	difficult	closed	Coughing	vigrous	C-UA	yes	90	84	86	84	99	108	99	105	98	102	96	97	96	97
16.	37026	26	54	165	M	I	II	17	EASY	ABDUCT	NONE	NONE	CA	No	91	102	91	94	96	114	96	111	94	109	92	102	90	98
17.	36857	35	50	167	M	II	I	13	EASY	ABDUCT	NONE	NONE	CA	No	92	98	84	89	94	120	94	113	92	106	90	103	88	99
18.	39131	21	52	162	M	I	I	19	difficult	closed	Coughing	vigrous	C-UA	yes	84	97	80	96	90	115	90	111	88	108	86	108	85	102
19.	37075	23	50	153	F	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	88	97	88	96	93	116	92	112	91	106	89	104	87	98
20.	37471	23	54	167	M	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	88	97	88	99	93	116	90	112	88	107	88	104	86	97
21.	57252	45	50	165	M	I	I	16	difficult	closed	Coughing	vigrous	C-UA	yes	85	98	85	98	100	120	100	112	98	109	97	104	95	103
22.	36856	28	55	151	F	I	I	14	difficult	closed	Coughing	vigrous	C-UA	No	90	97	90	97	93	120	93	113	91	109	91	103	88	100
23.	37650	50	56	164	M	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	85	98	84	90	90	115	90	112	88	108	86	105	84	100
24.	37512	40	52	163	M	I	I	17	difficult	closed	Coughing	vigrous	C-UA	yes	88	103	88	99	94	119	93	115	91	111	89	110	87	106
25.	37968	24	56	165	M	II	I	14	EASY	ABDUCT	Coughing	NONE	C-UA	No	91	97	85	97	97	118	97	118	94	118	92	110	90	106
26.	38609	42	49	166	M	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	90	97	90	97	99	119	99	117	93	111	91	109	90	105
27.	38757	46	54	167	M	I	I	18	difficult	closed	Coughing	vigrous	C-UA	No	85	98	83	96	85	115	85	111	83	109	81	103	79	101
28.	38578	38	57	160	M	I	I	18	EASY	ABDUCT	NONE	NONE	CA	No	100	90	91	89	93	111	93	106	90	103	88	97	86	95
29.	38581	45	50	164	M	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	102	85	93	84	95	106	95	102	93	100	91	96	89	94
30.	44707	35	52	153	F	I	I	14	difficult	closed	Coughing	vigrous	C-UA	No	96	97	96	96	100	113	99	110	97	108	95	103	93	100
31.	33077	47	65	161	M	I	I	12	EASY	ABDUCT	Coughing	NONE	C-UA	No	85	98	85	98	100	120	100	112	98	109	97	104	95	103
32.	33766	37	51	151	M	I	I	14	difficult	closed	Coughing	vigrous	C-UA	Yes	90	97	90	97	93	120	93	113	91	109	91	103	88	100
33.	35050	40	58	162	M	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	85	98	84	90	90	115	90	112	88	108	86	105	84	100
34.	32981	33	59	164	F	I	I	17	difficult	closed	Coughing	vigrous	C-UA	No	88	103	88	99	94	119	93	115	91	111	89	110	87	106
35.	33241	35	60	166	M	I	I	18	EASY	ABDUCT	NONE	NONE	CA	No	91	97	91	97	97	118	97	118	94	118	92	110	90	106
36.	33090	35	51	167	F	I	I	19	EASY	ABDUCT	NONE	NONE	CA	No	90	97	92	97	99	119	99	117	93	111	91	109	90	105
37.	33148	22	50	165	F	I	I	17	difficult	closed	Coughing	vigrous	C-UA	No	85	98	83	96	85	115	85	111	83	109	81	103	79	101
38.	33060	38	35	156	M	II	I	16	EASY	ABDUCT	NONE	NONE	CA	No	100	90	91	89	93	111	93	106	90	103	88	97	86	95
39.	31874	47	45	152	M	I	I	16	EASY	ABDUCT	NONE	NONE	CA	No	102	85	93	84	95	106	95	102	93	100	91	96	89	94
40.	32722	20	46	165	M	I	I	17	difficult	closed	Coughing	vigrous	C-UA	Yes	96	97	97	96	100	113	100	110	97	108	95	103	93	100
41.	33977	38	50	168	M	I	I	16	difficult	closed	Coughing	vigrous	C-UA	No	85	98	87	98	100	120	100	112	98	109	97	104	95	103
42.	33966	47	52	162	M	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	90	97	90	97	93	120	93	113	91	109	91	103	88	100
43.	34034	41	59	165	M	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	85	98	84	90	90	115	90	112	88	108	86	105	84	100
44.	33423	26	62	167	M	I	I	17	difficult	closed	Coughing	vigrous	C-UA	No	88	103	90	99	94	119	93	115	91	111	89	110	87	106
45.	33401	40	46	151	F	II	I	14	EASY	ABDUCT	NONE	NONE	CA	No	91	97	95	97	97	118	98	118	94	118	92	110	90	106
46.	32700	34	58	156	M	I	I	15	EASY	ABDUCT	NONE	NONE	CA	No	90	97	92	97	99	119	99	117	93	111	91	109	90	105
47.	32981	40	52	149	F	I	I	18	EASY	ABDUCT	NONE	NONE	CA	No	85	98	83	96	85	115	85	111	83	109	81	103	79	101
48.	32241	40	52	165	M	I	I	18	difficult	closed	Coughing	vigrous	C-UA	yes	100	90	91	89	93	111	93	106	90	103	88	97	86	95
49.	33090	25	46	148	F	I	I	12	EASY	ABDUCT	NONE	NONE	CA	No	102	85	93	84	95	106	95	102	93	100	91	96	89	94
50.	33,148	35	48	150	F	I	I	14	EASY	ABDUCT	NONE	NONE	CA	No	96	97	97	96	100	113	99	110	97	108	95	103	93	100

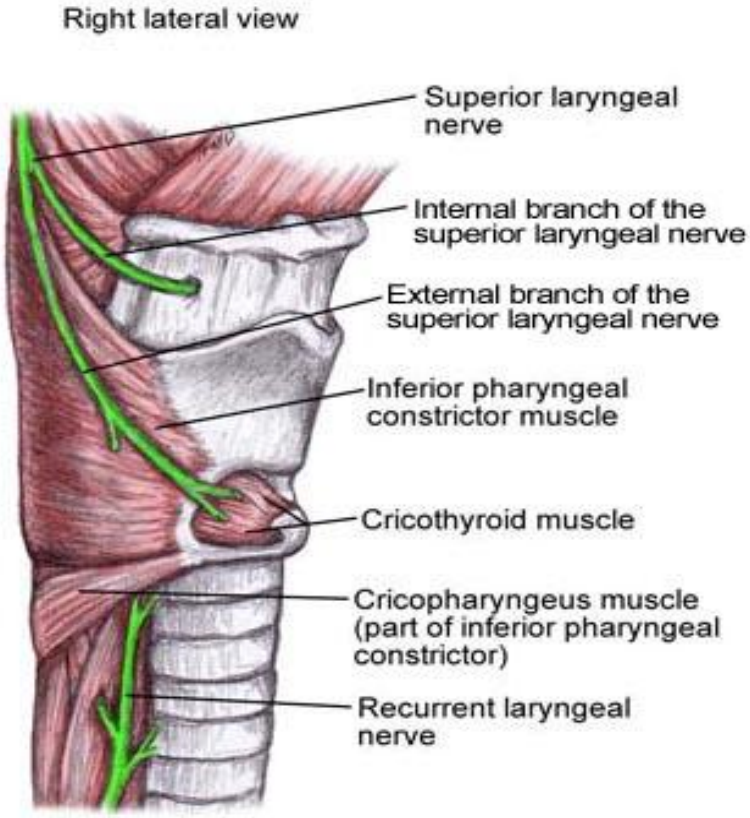
LARYNX ANATOMY



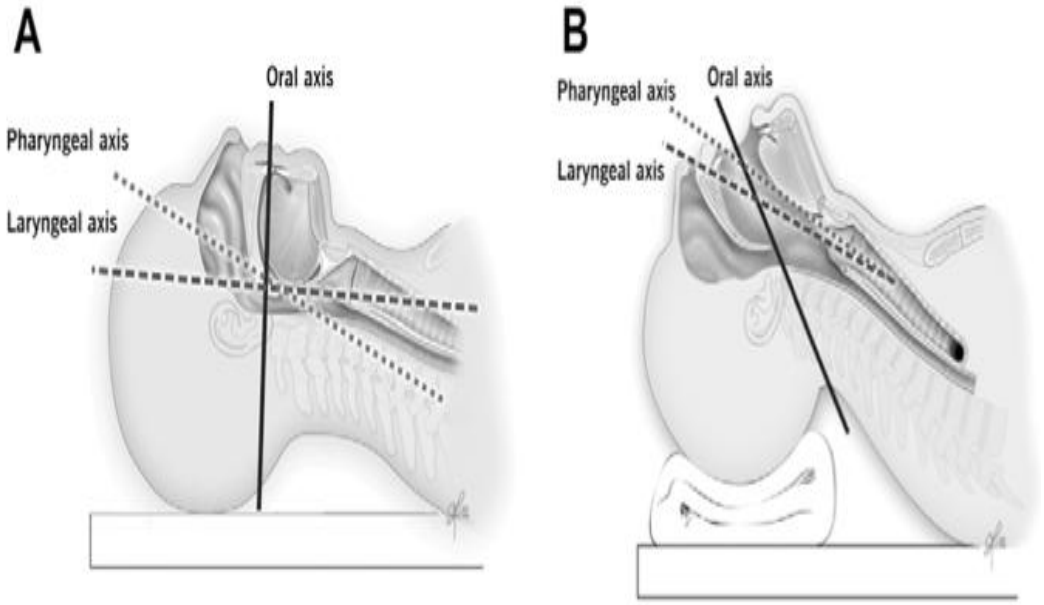
MUSCLES OF LARYNX



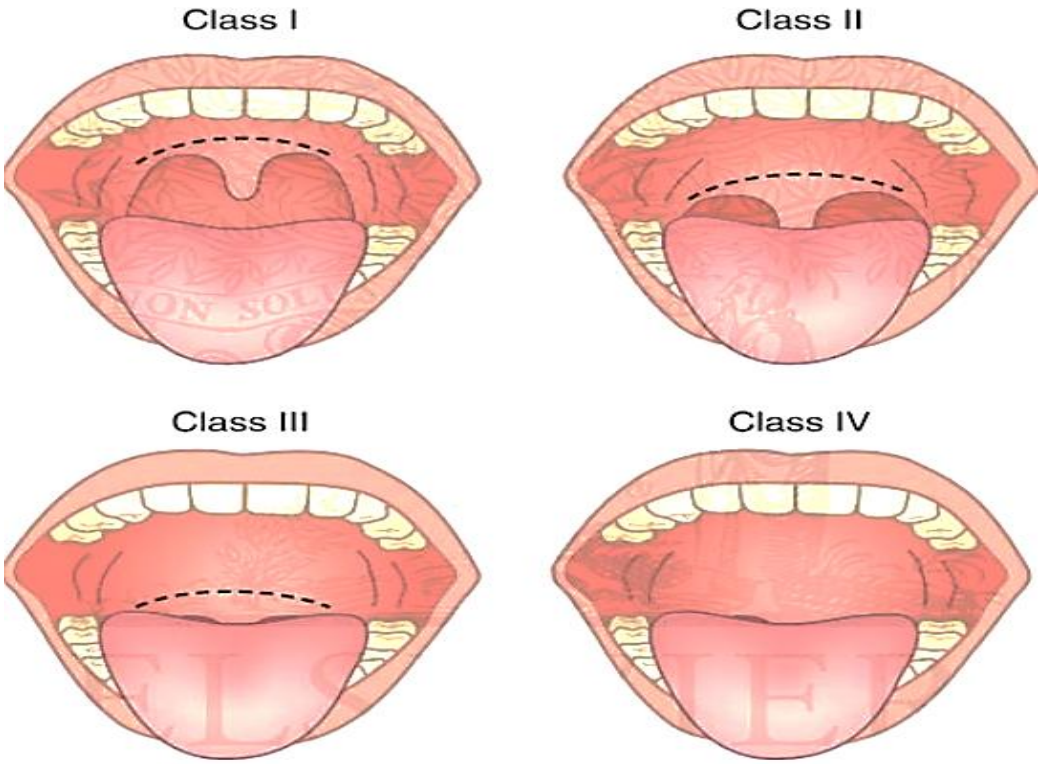
NERVE SUPPLY OF LARYNX



LARYNGEAL AXIS

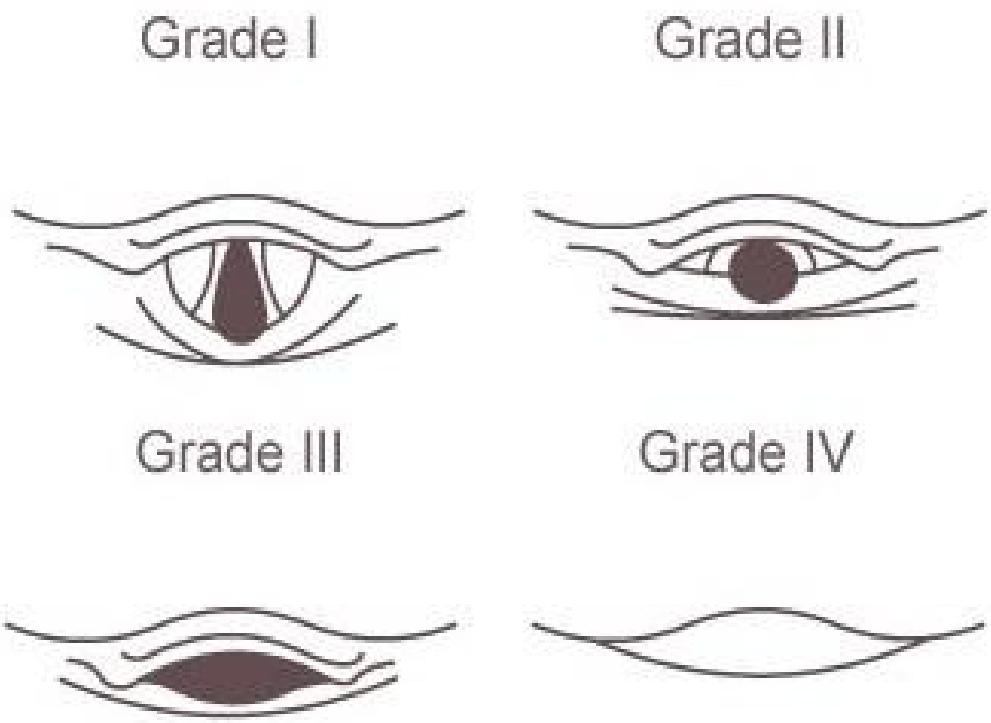


MALLAM PATTI CLASSIFICATION



CORMACK AND LEHANE GRADING OF

LARYNGOSCOPIC VIEW




10ml
PROPOFOL INJECTION
For Intravenous Use Only
Warning : To be given on the prescription of an Anaesthesiologist attached to hospitals, Institutions & nursing homes only. Contains no antimicrobial agents.
Manufactured in India by:


Lignocaine Hydrochloride
Injection IP
Xylocard®
जाइलोकार्ड
2%
50 ml
I.V.


5 ml
Midazolam Injection B.P.
5mg/5ml Sterile Solution
for IM/IV Use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